VIRGINIA MEDICAL MONTHLY

OFFICIAL PUBLICATION OF THE MEDICAL SOCIETY OF VIRGINIA

JUNE, 1960

FROM PARKE-DAVIS...A NEW ORAL ANTIBIOTIC

WITH EFFECTIVE ANTIBACTERIAL & ANTIAMEBIC ACTIONS

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ADMINISTRATION AND DOSAGE

(expressed in terms of base)

Condition	Dosage (daily, in divided doses)	Duration of Therapy
Infectious Diarrheas (bacillary and nonspecific)	*Adults:* 35 to 50 mg/Kg; up to 100 mg/Kg in severe cases.	Up to 7 days
	Children: 50 mg/Kg; up to 100 mg/Kg in severe cases.	Up to 7 days
Intestinal Amebiasis (acute, subacute, chronic)	*Adults:* 0.75 to 1.5 Gm.; larger doses when required.	5 days
	Children: 22 mg/Kg; larger doses when required.	5 days
Preoperative Suppression of Intestinal Flora	*Adults:* 2 Gm.	4 days
Hepatic Coma	*Adults:* up to 6 Gm., depending on degree of hepatic insufficiency and response of patient.	See literature

SIDE EFFECTS: Since HUMATIN by the oral route is virtually nonabsorbed in the gastrointestinal tract, even with exceptionally high doses, systemic toxicity has not been a problem in clinical use.[2,3,5-8] However, when doses in excess of 2 Gm. per day are given for more than three days, loose stools may develop; on doses of from 4 to 6 Gm. daily, moderately severe diarrhea has been reported in some patients. No other indications of toxicity have been observed.

SUPPLIED: HUMATIN (paromomycin, Parke-Davis) is available as the sulfate in Kapseals,® each containing 250 mg. of base, in bottles of 16. Literature supplying details of dosage and administration available on request.

REFERENCES: (1) Coffey, G. L., *et al.*: *Antibiotics & Chemother.* 9:730, 1959. (2) Personal Communications to the Department of Clinical Investigation, Parke, Davis & Company, 1959. (3) Godenne, G. D.: Paromomycin in diarrheas of infants and children, Antibiotics Annual 1959-1960, New York, Medical Encyclopedia Inc., in press. (4) McMath, W. F. T., & Hussain, K. K.: *Pub. Health* 73:328, 1959. (5) Courtney, K. O., & Thompson, P. E.: Paromomycin as a therapeutic substance for intestinal amebiasis and bacterial enteritis, Antibiotics Annual 1959-1960, New York, Medical Encyclopedia Inc., in press. (6) Shafei, A. Z.: *Antibiotic Med. & Clin. Therapy* 6:275, 1959. (7) Elias, F. L., & Oliver-Gonzales, J.: *Antibiotic Med. & Clin. Therapy* 6:584, 1959. (8) Carter, C. H.: *Antibiotic Med. & Clin. Therapy* 6:586, 1959. (9) Fast, B. B., *et al.*: *Arch. Int. Med.* 101:467, 1958. (10) Mackie, J. E., *et al.*: *New England J. Med.* 259:1151, 1958. (11) Stormont, J. M., *et al.*: *New England J. Med.* 259:1145, 1958.

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TABLE OF CONTENTS

GUEST EDITORIAL
This I Believe—*Lamont Pugh, M.D.* 297

ORIGINAL ARTICLES
Plant Dermatitis—*George H. Curtis, M.D.* 301
The Educational Work of Robley Dunglison, M.D.—*Chalmers L. Gemmill, M.D.* 307
The Control of Weight in Hypertensives on Rauwolfia and its Derivatives—*Harold S. Feldman, M.D.* 310
Portal Caval Shunt—*R. N. deNiord, Jr., M.D.* 314
Immuno-Pancreatitis—*Robert Edgar Mitchell, Jr., M.D., James L. A. Roth, M.D., H. L. Bockus, M.D., Merle M. Miller, M.D.* 319
The Problem of Hiatus Hernia—*William D. Poe, M.D.* ... 324
Aspirin Poisoning—$C_9H_8O_4$—*Sidney Kaye, Ph.D.* 330
Tobacco and Renal Function and Diseases of the Urinary Tract—*P. S. Larson, Ph.D., H. B. Haag, M.D., H. Silvette, Ph.D.* 332

PRE-PAID MEDICAL CARE
The Past Five Years 342

PUBLIC HEALTH
Control of Communicable Diseases in Man 344

MENTAL HEALTH
Psychiatric Consultation to Social Agencies 347

THE MEDICAL SOCIETY OF VIRGINIA
Council Minutes 349

MISCELLANEOUS
Mail-Order Rx Schemes 351

BOOK ANNOUNCEMENTS 353

EDITORIAL
The University of Virginia Hospital (1901-1951)—*Harry J. Warthen, M.D.* 354

SOCIETY ACTIVITIES 355

NEWS 356

OBITUARIES 357

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INDEX TO ADVERTISERS—Page 80

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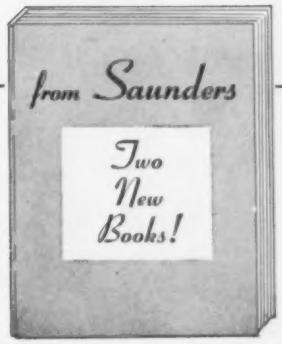

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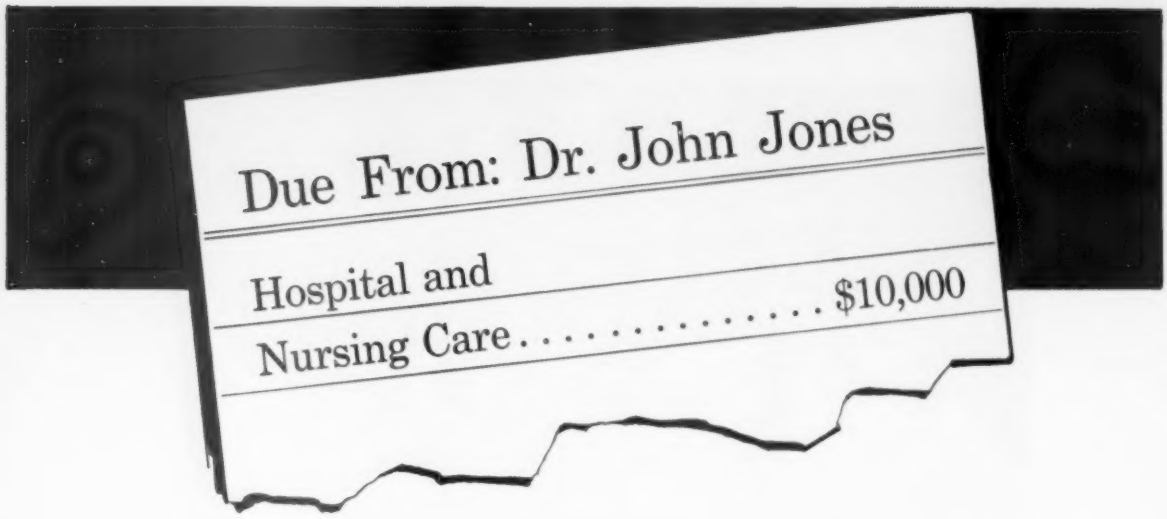

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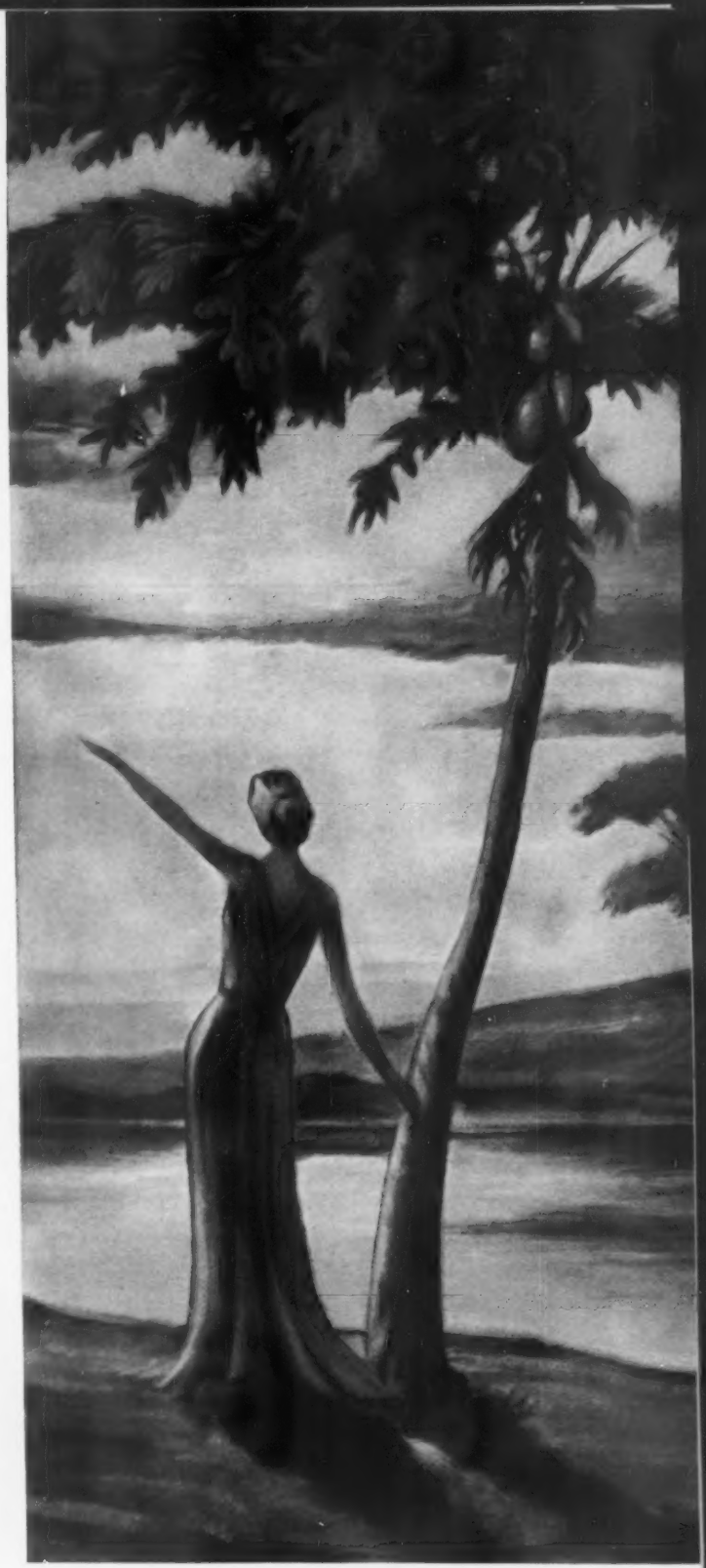

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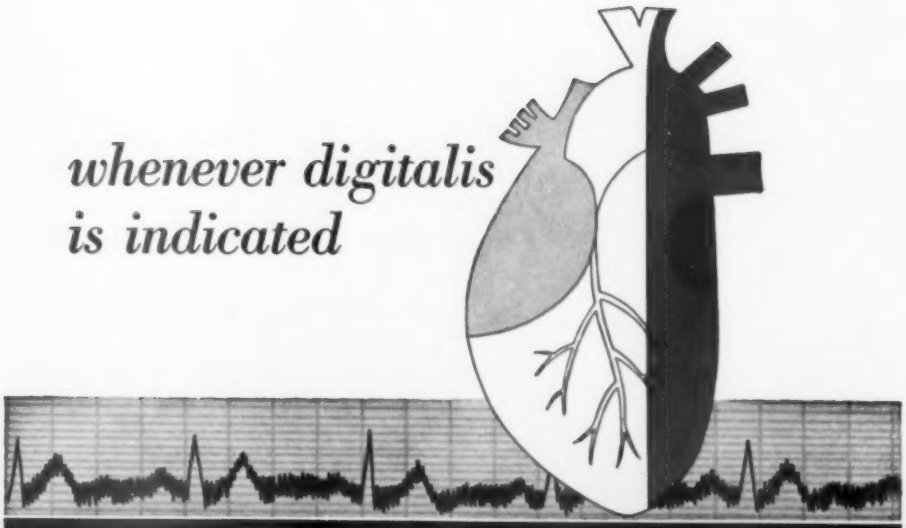

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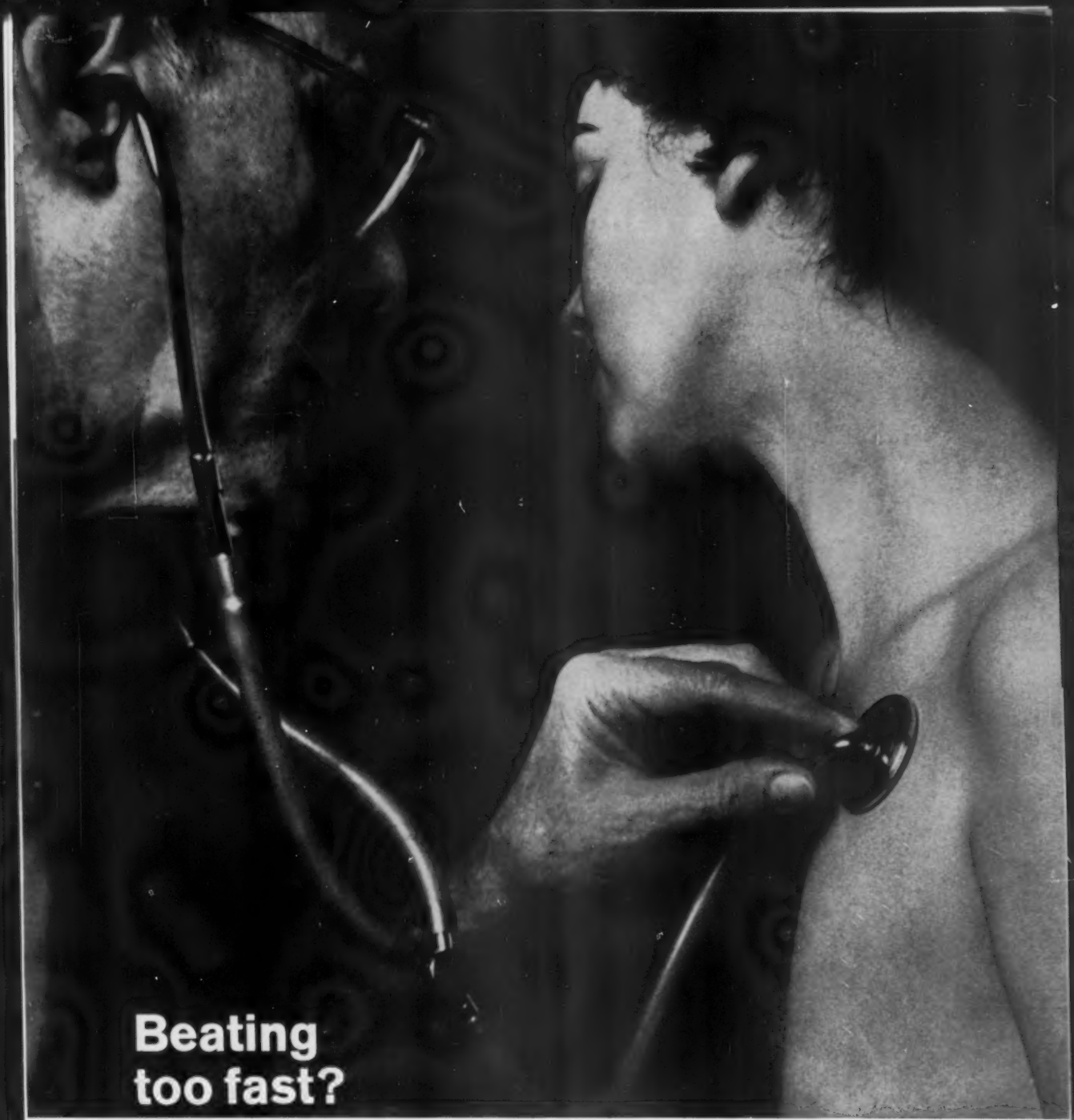


Beating too fast? Slow it down with SERPASIL®

(reserpine CIBA)

Serpasil has proved effective as a heart-slowing agent in the following conditions: mitral disease; myocardial infarction; cardiac arrhythmias; neurocirculatory asthenia; thyroid toxicosis; excitement and effort syndromes; cardiac neurosis; congestive failure. Serpasil should be used with caution in patients receiving digitalis and quinidine. It is not indicated in cases of aortic insufficiency.

SUPPLIED: Tablets, 0.1 mg., 0.25 mg. (scored) and 1 mg. (scored). Complete information available on request.

CIBA
SUMMIT•NEW JERSEY

[illegible]

control the tension–treat the trauma

...Pathibamate® 400 200

meprobamate with PATHILON® tridihexethyl chloride Lederle

greater flexibility in the control of tension, hypermotility and excessive secretion in gastrointestinal dysfunctions

PATHIBAMATE combines two highly effective and well-tolerated therapeutic agents:

meprobamate (400 mg. or 200 mg.) widely accepted tranquilizer and ...
PATHILON (25 mg.)—anticholinergic noted for its peripheral, atropine-like action, with few side effects.

The clinical advantages of PATHIBAMATE have been confirmed by nearly two years' experience in the treatment of duodenal ulcer; gastric ulcer; intestinal colic; spastic and irritable colon; ileitis; esophageal spasm; anxiety neurosis with gastrointestinal symptoms and gastric hypermotility.

Two dosage strengths—PATHIBAMATE-400 and PATHIBAMATE-200 facilitate individualization of treatment in respect to both the degree of tension and associated G.I. sequelae, as well as the response of different patients to the component drugs.

Supplied: **PATHIBAMATE-400**—Each tablet (yellow, ½-scored) contains meprobamate, 400 mg.; PATHILON tridihexethyl chloride, 25 mg.
PATHIBAMATE-200—Each tablet (yellow, coated) contains meprobamate, 200 mg.; PATHILON tridihexethyl chloride, 25 mg.

Administration and Dosage: **PATHIBAMATE-400**—1 tablet three times a day at mealtime and 2 tablets at bedtime.
PATHIBAMATE-200—1 or 2 tablets three times a day at mealtime and 2 tablets at bedtime.
Adjust to patient response.

Contraindications: glaucoma; pyloric obstruction, and obstruction of the urinary bladder neck.

Lederle LEDERLE LABORATORIES, A Division of AMERICAN CYANAMID COMPANY, Pearl River, New York

Maximal Absorption
Acid stable, highly soluble

Maximal Blood Levels

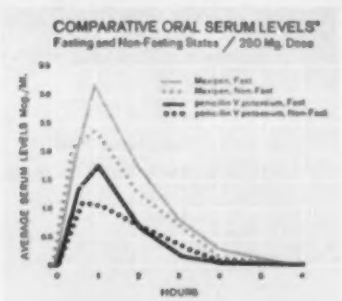
Maximal Flexibility
May be administered without regard to meals. However, highest absorption is achieved when taken just before or between meals.

Maximal Oral Indications
Indicated in infections caused by streptococci, pneumococci, susceptible staphylococci, and gonococci

DOSAGE: For moderately severe conditions, 125 to 250 mg. three times daily. For more severe conditions, 500 mg. as often as every four hours around the clock.

NOTE: To date, MAXIPEN has not shown less allergic reactions than older oral penicillins. Usual precautions regarding penicillin administration should be observed.

SUPPLIED: MAXIPEN TABLETS, scored, 125 mg. (200,000 units), bottles of 36; 250 mg. (400,000 units), bottles of 24 and 100 tablets. MAXIPEN FOR ORAL SOLUTION; reconstituted each 5 cc. contains 125 mg. (200,000 units), in 60 cc. bottles.



*Based on 3294 individual serum antibiotic determinations. Complete details available on request.

MAXIPEN, the orally maximal penicillin, is a triumph of man over molecule; a product of Pfizer Research

ROERIG Rx
New York 17, N. Y.
Division, Chas. Pfizer & Co., Inc.
Science for the World's Well-Being

You see an improvement within a few days
Thanks to your prompt treatment and the smooth action of Deprol, her depression is relieved and her anxiety and tension calmed – *often in a few days.* She eats well, sleeps well and soon returns to her normal activities.

Lifts depression...as it calms anxiety!

Smooth, balanced action lifts depression as it calms anxiety...rapidly and safely

Balances the mood – no "seesaw" effect of amphetamine-barbiturates and energizers. While amphetamines and energizers may stimulate the patient – *they often aggravate anxiety and tension.*

And although amphetamine-barbiturate combinations may counteract excessive stimulation – *they often deepen depression.*

In contrast to such "seesaw" effects, Deprol's smooth, *balanced* action lifts depression as it calms anxiety – both at the same time.

Acts swiftly – the patient often feels better, sleeps better, within a few days. Unlike the delayed action of most other antidepressant drugs, which may take two to six weeks to bring results, Deprol relieves the patient quickly – often within a few days. Thus, the expense to the patient of long-term drug therapy can be avoided.

Acts safely – no danger of liver damage. Deprol does not produce liver damage, hypotension, psychotic reactions or changes in sexual function – frequently reported with other antidepressant drugs.

Dosage: Usual starting dose is 1 tablet q.i.d. When necessary, this dose may be gradually increased up to 3 tablets q.i.d.

Composition: 1 mg. 2-diethylaminoethyl benzilate hydrochloride (benactyzine HCl) and 400 mg. meprobamate. **Supplied:** Bottles of 50 light-pink, scored tablets. Write for literature and samples.

▲Deprol▲®

WALLACE LABORATORIES/*New Brunswick, N. J.*

69-3735

THIS IS
THE
TABLET

ALPEN is the oral penicillin that provides on a fasting stomach peak antibiotic blood levels approximately twice as high as oral potassium penicillin V... and significantly higher than I. M. penicillin G.

Some strains of staphylococci resistant to other penicillins exhibit in vitro sensitivity to potassium phenethicillin.

ALPEN has greater freedom from the G. I. sequelae (overgrowth of resistant flora) sometimes observed with broad spectrum -mycins.

ALPEN gives much higher antibiotic levels within the first hour of ingestion by the well-tolerated oral route.

WHEN TO USE ALPEN Recommended in the treatment of infections caused by pneumococci, streptococci, gonococci, corynebacteria, and penicillin-sensitive staphylococci.

HOW TO USE ALPEN Depending on the severity of the infection, 125 mg. (200,000 units) or 250 mg. (400,000 units) three times daily may be used. In more severe or stubborn infections, a dosage of 500 mg. (800,000 units) t.i.d. may be employed. In beta hemolytic streptococcal infections, treatment should be continued for at least ten days.

PRECAUTIONS The usual precautions in the administration of oral penicillin should be observed. For further details see package literature.

Tablets: 125 mg. and 250 mg., bottles of 25 and 100. Powder for Oral Solution (lemon-lime flavored), 1.5 Gm. bottle (125 mg. per 5 cc. teaspoonful).

this is the tablet that gives higher peak antibiotic blood levels

HIGHER THAN I. M. PENICILLIN G
HIGHER THAN POTASSIUM PENICILLIN V

ALPEN

ALPEN™–potassium phenethicillin

Schering

for adults and children

Prompt 4 way check of diarrhea

- Curbs excessive peristalsis
- Adsorbs toxins and gases
- Soothes inflamed mucosa
- Provides intestinal antisepsis

POMALIN

TRADEMARK

Liquid

EFFECTIVE ANTIDIARRHEAL

FORMULA: *Each 15 cc. (tablespoon) contains:*

Sulfaguanidine	2 Gm.
Pectin	225 mg.
Kaolin	3 Gm.
Opium tincture	0.08 cc.
(equivalent to 2 cc. paregoric)	

DOSAGE: **Adults:** Initially 1 or 2 tablespoons from four to six times daily, or 1 or 2 teaspoons after each loose bowel movement; reduce dosage as diarrhea subsides.

Children: ½ teaspoon (=2.5 cc.) per 15 lb. of body weight every four hours day and night until stools are reduced to five daily, then every eight hours for three days.

SUPPLIED: *Bottles of 16 fl. oz. (raspberry flavor, pink color)*
Exempt Narcotic. Available on Prescription Only.

Winthrop LABORATORIES
New York 18, N. Y.

the right combination

. . . safe, effective antihypertensive therapy

Rauprote

(Rauwolfia serpentina and Protoveratrines A and B combined)

Rauprote combines two effective antihypertensive agents—*Rauwolfia serpentina* for moderate tranquillizing and gentle hypotensive effect, and Protoveratrines A and B for faster, more potent lowering of blood pressure and bradycrotic action. The combination produces a therapeutic hypotensive effect which is superior to larger doses of either drug alone; reduced dosage of both components minimizes or eliminates toxic side effects completely.[1,2]

Clinical studies show the majority of patients suffering from significant elevation of blood pressure achieve an excellent response to this combination.[2]

Rauprote is indicated in management of moderate to severe essential hypertension.

Supplied:

In bottles of 100 and 1,000 tablets, each tablet containing 50 mg. *Rauwolfia serpentina* and 0.2 mg. Protoveratrines A and B (alkaloids of *Veratrum album*).

1. Goodman, L.S. and Gilman, A.: The Pharmacological Basis of Therapeutics, 2nd Ed., Macmillan & Co., New York (1955).

2. Roberts, E.: Four Year Evaluation of an Antihypertensive Agent, J. Am. Med. Women's Assn. *13*:349 (1958).

THE VALE CHEMICAL CO., INC.

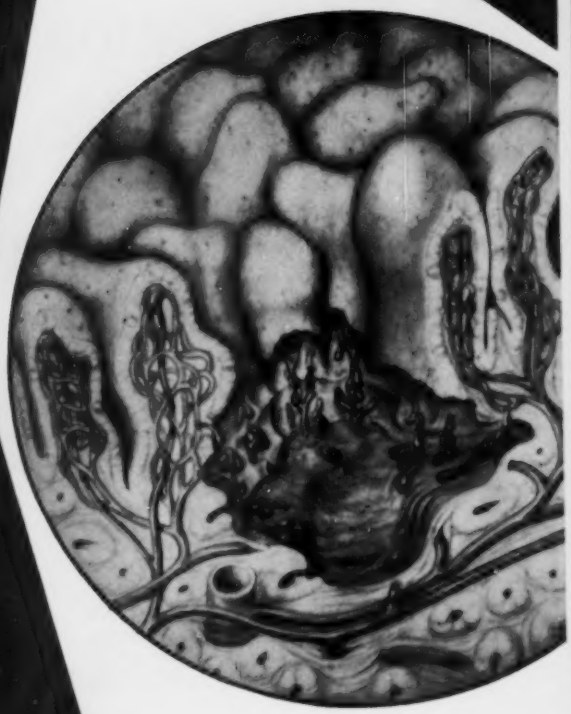
VALE

Pharmaceuticals since 1922

Allentown, Pennsylvania

strengthens
fragile capillaries

in internal bleeding



capillary hemorrhage
in duodenal ulcer

. . . associated with abnormal capillary permeability and fragility in

peptic ulcer

ulcerative colitis

chronic nosebleed

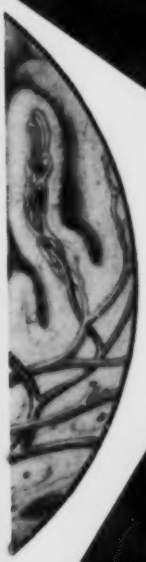
purpura
(nonthrombocytopenic)

hemorrhagic cystitis

ecchymoses

menorrhagia

habitual and threatened abortion



CVP®

C.V.P. helps diminish increased capillary permeability, fragility, and resultant bleeding by acting to maintain the integrity of the intercellular ground substance (cement) of capillary walls. C.V.P. is the original and exclusive water-soluble citrus bioflavonoid complex. Readily absorbed and utilized, C.V.P. is relatively free (due to special processing) of hesperidin, naringin and other comparatively insoluble and inactive flavonoids found in citrus.

Each capsule or teaspoonful (approx. 5 cc.) of syrup provides:

CITRUS BIOFLAVONOID COMPOUND	100 mg.
ASCORBIC ACID (vitamin C)	100 mg.

Bottles of 100, 500 and 1000 capsules; 4 oz., 16 oz. and gallon syrup

samples (capsules or syrup) and literature from . . .

u. s. vitamin corporation • PHARMACEUTICALS

(Arlington-Funk Laboratories, division) • 250 East 43rd Street, New York 17, N. Y.

now–for more comprehensive control of *'pain & spasm'*

INDICATIONS

HEAD: temporomandibular muscle spasm • NECK: acute torticollis, osteoarthritis of cervical spine with spasm of cervical muscles, whiplash injury • TRUNK AND CHEST: costochondritis, intercostal myositis, xiphodynia • BACK: acute and chronic lumbar strains and sprains, acute low back pain (unspecified), acute lumbar arthritis and traumatic injury, compression fracture, herniated intervertebral disc, post-disc syndrome, strained muscle(s) • EXTREMITIES: acute hip injury with muscle spasm, ankle sprain, arthritis (as of foot or knee), blow to shin followed by muscle spasm, bursitis, spasm or strain of muscle or muscle group, old fracture with recurrent spasm, Pellegrini-Stieda disease, tenosynovitis with associated pain and spasm.

-pain due to
or associated with
-spasm of skeletal muscle

a new muscle relaxant-analgesic

Robaxisal

ROBAXIN® WITH ASPIRIN

Many conditions, painful in themselves, often give rise to spasm of skeletal muscles. ROBAXISAL, the new dual-acting muscle relaxant-analgesic, treats both the pain and the spasm with marked success! In clinical studies on 311 patients, 12 investigators[1] reported satisfactory results in 86.5%. Each ROBAXISAL Tablet contains:

- A relaxant component — Robaxin® — widely recognized for its prompt, long-lasting relief of painful skeletal muscle spasm, with unusual freedom from undesired side effects . . . . . 400 mg.

  ®Methocarbamol Robins U.S. Pat. No. 2770569

- An analgesic component — aspirin — whose pain-relieving effect is markedly enhanced by Robaxin, and which has added value as an anti-inflammatory and anti-rheumatic agent. . . . (5 gr.) 325 mg.

INDICATIONS: ROBAXISAL is indicated when analgesic as well as relaxant action is desired in the treatment of skeletal muscle spasm and severe concurrent pain. Typical conditions are disorders of the back, whiplash and other traumatic injuries, myositis, and pain and spasm associated with arthritis.

SUPPLY: ROBAXISAL Tablets (pink-and-white, laminated) in bottles of 100 and 500.

Also available: ROBAXIN Injectable, 1.0 Gm. in 10-cc. ampul. ROBAXIN Tablets, 0.5 Gm. (white, scored) in bottles of 50 and 500.

[1]Clinical reports in files of A. H. Robins Co., Inc., from: J. Allen, Madison, Wisc., B. Billow, New York, N. Y., E. Decker, Richmond, Va., C. Freeman, Jr., Augusta, Ga., R. F. Gordon, New York, N. Y., J. E. Heimbold, Schenectady, N. Y., L. Levy, New York, N. Y., N. Loliee, Chicago Heights, Ill., H. Mackenna, Richmond, Va., A. Poundstone, Los Angeles, Cal., E. Rogers, Brooklyn, N. Y., K. H. Strong, Fairfield, Ia.

Additional information available upon request.

Robins A. H. ROBINS CO., INC., Richmond 20, Virginia

Making today's medicines with integrity . . . seeking tomorrow's with persistence

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Recognizing that the exchange of ideas is fundamental to medical progress, Lederle continues its Symposium program with the 9th year of scheduled meetings. Through these Symposia, sponsored by medical organizations with our cooperation, over 50,000 physicians have had the opportunity to hear and question authorities on important advances in clinical medicine and surgery. You have a standing invitation to attend any of these Symposia with your wife, for whom a special program is planned.

ANCHORAGE, ALASKA
Saturday, June 11, 1960
The Westward Hotel

WEST POINT, NEW YORK
Thursday, Friday, Saturday,
June 16, 17, and 18, 1960
United States Thayer Hotel

***MADISON, WISCONSIN**
Thursday, June 23, 1960
The Holiday Inn

***SPRINGFIELD, MISSOURI**
Sunday, June 26, 1960
The Holiday Inn

***ROANOKE, VIRGINIA**
Saturday, July 16, 1960
The Hotel Roanoke

***SANTA ROSA, CALIFORNIA**
Friday, September 16, 1960
The Flamingo Hotel

***KANSAS CITY, KANSAS**
Friday, September 23, 1960
Battenfeld Memorial
Auditorium

HOUSTON, TEXAS
Saturday, September 24, 1960
The Shamrock Hilton Hotel

DEFIANCE, OHIO
Wed., September 28, 1960
Defiance College

PHILADELPHIA, PENN.
Sunday, October 16, 1960
The Sheraton Hotel

***HARTFORD, CONNECTICUT**
Thursday, October 20, 1960
The Statler Hotel

***GREAT FALLS, MONTANA**
Saturday, October 22, 1960
The Rainbow Hotel

ROCHESTER, NEW YORK
Wednesday, October 26, 1960
The Manger Hotel

CHARLESTON, WEST VIRGINIA
Sunday, October 30, 1960
The Daniel Boone Hotel

SIOUX FALLS, SOUTH DAKOTA
Tuesday, November 1, 1960
The Sheraton-Cataract Hotel

***CHARLOTTE, N. CAROLINA**
Thursday, November 3, 1960
The Hotel Charlotte

***CLEVELAND, OHIO**
Wednesday, November 9, 1960
Pick Carter Hotel

***SOUTH BEND, INDIANA**
Friday, November 18, 1960
The Pick-Oliver Hotel

WESTCHESTER COUNTY, N. Y.
Wednesday November 30, 1960
Westchester Country Club

ST. PETERSBURG, FLORIDA
Saturday, December 3, 1960
Tides Hotel and Bath Club

*Acceptable for Category I Credit for members of American Academy of General Practice

Lederle **LEDERLE LABORATORIES, a Division of AMERICAN CYANAMID COMPANY, Pearl River, N. Y.**

Thanks to your prompt treatment and the smooth action of Deprol, your patient's depression is relieved and her anxiety calmed—*often in two or three days.* She eats properly, sleeps well, and her depression no longer complicates your basic regimen.

Lifts depression...as it calms anxiety!

For pregnant, postpartum and menopausal patients—a smooth, balanced action that lifts depression as it calms anxiety...rapidly and safely

Balances the mood—no "seesaw" effect of amphetamine-barbiturates and energizers. While amphetamines and energizers may stimulate the patient—*they often aggravate anxiety and tension.* And although amphetamine-barbiturate combinations may counteract excessive stimulation—*they often deepen depression.*

In contrast to such "seesaw" effects, Deprol lifts depression as it calms anxiety.

Acts swiftly—the patient often feels better, sleeps better, within two or three days. Unlike most other antidepressant drugs, Deprol relieves the patient quickly—often within two or three days.

Acts safely—no psychotic reactions. Deprol does not cause hypotension, tachycardia, jitteriness, or liver toxicity. It can be safely administered with basic therapy.

Dosage: Usual starting dose is 1 tablet q.i.d. When necessary, this may be gradually increased up to 3 tablets q.i.d.
Composition: 1 mg. 2-diethylaminoethyl benzilate hydrochloride (benactyzine HCl) and 400 mg. meprobamate.
Supplied: Bottles of 50-light-pink, scored tablets. Write for literature and samples.

Deprol

WALLACE LABORATORIES
New Brunswick, N. J.

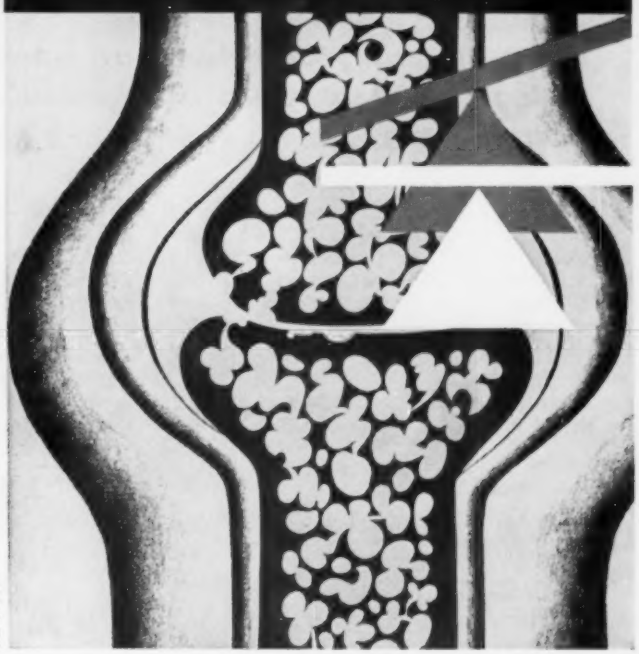
CD-709

Sterazolidin®

brand of prednisone-phenylbutazone

a well balanced therapy in all forms of rheumatic disorder

for rapid, effective relief



The combined action of phenylbutazone and prednisone in Sterazolidin results in striking therapeutic benefit with only moderate dosage of both active agents.

In long-term therapy of the major forms of arthritis, control is generally maintained indefinitely with stable uniform dosage safely below that likely to produce significant hypercortisonism.

In short-term therapy of more acute conditions Sterazolidin provides intensive anti-inflammatory action to assure early resolution and recovery.

Sterazolidin®, brand of prednisone-phenylbutazone: Each capsule contains prednisone, 1.25 mg.; Butazolidin® (brand of phenylbutazone), 50 mg.; dried aluminum hydroxide gel, 100 mg.; magnesium trisilicate, 150 mg.; homatropine methylbromide, 1.25 mg. Bottles of 100.

Geigy, Ardsley, New York

Geigy

SZ 1-60

Analgesic-Antipyretic-Sedative

Butapap®

A unique drug combination with wide clinical usefulness

Quite often, when children are administered an analgesic, a mild sedative is also indicated, to avoid the restlessness which frequently occurs as pain lessens.

In BUTAPAP, for the first time, this unique combination of drugs in easy-to-take liquid form provides a preparation that is highly useful wherever the allaying of pain or discomfort, fever, or restlessness is desired.

In BUTAPAP the potent analgesic effect of acetyl-p-aminophenol is potentiated by the inclusion of butabarbital sodium. The resultant effectiveness against pain and discomfort, and the unusual antipyretic action of acetyl-p-aminophenol, are reinforced by the sedative action of the butabarbital sodium, providing a preparation with wide clinical usefulness.

Each 5 cc. teaspoonful of tasty Butapap contains:

Butabarbital Sodium (¼ gr.) 15.0 mg.
Acetyl-p-aminophenol (2 gr.) 120.0 mg.

SAMPLES AND LITERATURE GLADLY SENT UPON REQUEST

CAUTION:
Federal law prohibits dispensing without prescription.

PHYSICIANS **PRODUCTS CO., INC.**
PETERSBURG, VIRGINIA

OUTSTANDING

to prevent
and clear up

diaper rash

DESITIN®
OINTMENT

physically Desitin Ointment assures constant protection against the irritation of urine and excrement.

bacteriostatically it markedly inhibits ammonia-producing bacteria.

therapeutically Desitin Ointment soothes, lubricates—and stimulates healing by means of high grade cod liver oil, rich in vitamins A and D and unsaturated fatty acids.

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DESITIN CHEMICAL COMPANY • 812 Branch Avenue, Providence 4, R. I.

announcing a major event in anticoagulant therapy...

Schering

Certified—before introduction—by 5 years of clinical experience and published reports in the U.S.A., Canada and Great Britain.

Miradon®

anisindione

new oral prothrombin depressant

control at every stage of anticoagulant therapy **rapidity** of induction and recovery time **predictability** of initial and maintenance dosages **stability** of therapeutic prothrombin levels during maintenance therapy **reversibility** of anticoagulant effect with vitamin K_1 preparations... rapid return to therapeutic levels on remedication

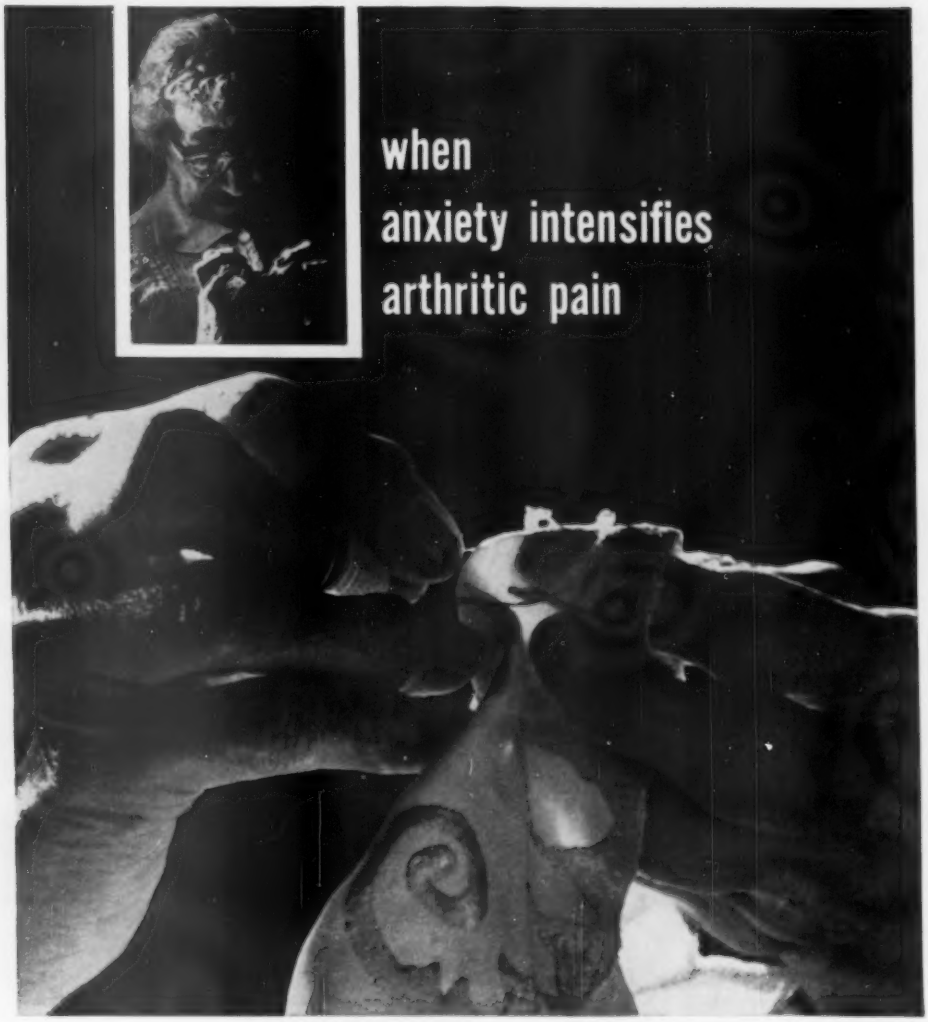
Well tolerated and relatively nontoxic no nausea and vomiting, proteinuria, agranulocytosis or leukopenia yet observed —chromaturia infrequent and transient.

Single daily dose convenience

Packaging—MIRADON Tablets, 50 mg., bottle of 100.

For complete information on indications, dosage, precautions, and contraindications consult the Schering Statement of Directions.

5-425



. . . DARVO-TRAN™ relieves pain more effectively than the analgesic components alone

Effective analgesia *plus* safe relief of mild anxiety helps combat the pain-anxiety spiral. In Darvo-Tran, the tranquilizing properties of Ultran® are added to the established analgesic effects of Darvon® and the anti-inflammatory benefits of A.S.A.®. Clinical and pharmacologic studies have shown that when pain is accompanied by anxiety, the addition of Ultran *enhances* and *prolongs* the analgesic effects of Darvon.

Lilly

Each Pulvule® Darvo-Tran provides:

Darvon	32 mg.	TO RAISE PAIN THRESHOLD
A.S.A.	325 mg.	TO REDUCE INFLAMMATION
Ultran	150 mg.	TO RELIEVE ANXIETY

Usual Dosage:
1 or 2 Pulvules three or four times daily.

Darvo-Tran™ (dextro propoxyphene and acetylsalicylic acid with phenaglycodol, Lilly)
Ultran® (phenaglycodol, Lilly)
Darvon® (dextro propoxyphene hydrochloride, Lilly)
A.S.A.® (acetylsalicylic acid, Lilly)

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The Virginia
MEDICAL
MONTHLY

June, 1960
VOL. 87, No. 6
Whole No. 1297

Guest Editorial . . . .

This I Believe

DR. JOHN CHALMERS DACOSTA used to caution older doctors on their way down the ladder not to interfere with the progress of younger members of the profession on their way up.

Mindful as I am of the meaningfulness of his admonition in that regard and possessed of an aversion to cluttering up the fresh air and sunshine of today with possibly musty ideas of yesteryear, it is in the face of considerable trepidation that I accept an invitation to write a guest editorial for the Virginia Medical Monthly.

In any event I was honored and flattered to have been considered worthy of such recognition as is inherent in Dr. Warthen's invitation.

In "Tom Jones," Henry Fielding observed that one who presumed to write should provide his potential readers with a declaration, in advance, of what he intended to record, similarly as a proprietor of a public eating place provides a bill of fare for his prospective customers. Having thus been notified the reader, like the customer, is free to either take or leave the offering.

It occurs to me that one who has been exposed to the various facets or disciplines of the practice of medicine for in excess of 40 years (if the time spent in medical school is counted) must inevitably be left with certain rather definite impressions. I believe I am so imbued and in compliance with the doctrine of Mr. Fielding I shall simply state it is my intention to set forth, in a manner that may be somewhat hodgepodge—but not purposely so—some tenets born of experience, some views that have appeared to become fixed with time and some convictions that have been engendered by my observations along the way. These comments will relate (a) to matters of a strictly clinical nature and (b) to the current status and needs of medicine as a profession. In fewer words; the bearing is initially clinical and then administrative.

Therefore, to the end that the element of orderliness in arrangement may not be completely disregarded, starting in the region of the head and proceeding caudalward: I believe that tonsils are provided for a definite physiological purpose, that of watchmen, so to speak, at the gateway to two important systems, the respiratory and the digestive, and their removal, for no reason other than their presence, is wrong. In substantiation of this view I would cite the relatively greater predisposition towards cervical adenopathy—in association with acute colds—on the part of those without tonsils in contrast to those with tonsils.

I believe there is a correlation between acute tonsillitis and acute appendicitis. Time and space will not permit me to elaborate upon my reasons for thinking thus and so. Suffice it, in any event, to say that during a three year tour of duty at the hospital of the U. S. Naval Academy at Annapolis, I had occasion to observe many Midshipmen

during epidemics of acute respiratory infections, and it was conspicuously notable that among this group (all of approximately the same age, of the same sex, and following the same routine with respect to diet, sleeping and exercise) with each rise in the incidence of acute tonsillitis, acute appendicitis also rose in a corresponding degree. It, at the same time, seems strikingly odd, however, that the two conditions existing concurrently in the same individual were not observed in a single instance.

I believe the manner of dress, particularly the protection of the upper chest and throat is important in the prevention of acute colds. At San Diego, California, the Naval Training Station and that of the U.S. Marine Corps are on adjacent compounds. Here again the ages of the recruits in the two service branches and their routines of training and of living were quite similar. Nevertheless, the hospital admission rate for Navy recruits with acute upper respiratory infections was from 50 to 75% higher than among Marine recruits. The major difference between the two groups was that the sailors wore jumpers open at the neck while the Marines wore shirts with buttoned up collars.

I believe that meningococcic meningitis is not contagious. In a careful and prolonged survey at the U.S. Naval Training Station, Great Lakes, Illinois, it was established that there was no space or proximity relationship between the numerous cases of spinal meningitis that developed in the barracks. Neither was there a relationship between those who showed positive meningococcus cultures from their naso-pharynges and those who subsequently developed meningitis. The incidence of meningitis among those who had previously shown positive cultures was no higher than among those with previously negative cultures.

I believe that cancer of the stomach, once it has become well enough defined to be macroscopically recognizable as such in the gross resected specimen, is about as hopeless of cure by any means as is a malignant melanoma. In my experience, all patients upon whom a diagnosis of cancer of the stomach had been correctly made preoperatively, no matter how early, and all of those in whom the existence of cancer was obvious at the time of operation, ultimately died of cancer, regardless of the extent to which surgery was carried, i.e., whether partial or total gastrectomy.

I believe peptic ulcer is a local manifestation of a systemic disease and that, as yet, no satisfactory explanation has been advanced for the occurrence of an open lesion per se any more than a plausible explanation has been offered for the formation of aphthous ulcers of the mouth, "fever blisters" on the lips or as to why a skin rash of one exanthematous disease shows a distribution and configuration characteristic of that disease while another is characterized by a rash of a different pattern.

I was taught in medical school—and for a long time was a devotee to the doctrine—that a perforated peptic ulcer was a surgical emergency of the first magnitude, and that unless closed by suture the patient would inevitably die. There was a time lapse schedule between perforation and closure according to which the prognosis was good, guarded, bad, grave or hopeless. I believe the validity of that teaching is subject to definite qualification. Undoubtedly many—in fact the majority—of perforations of gastric or duodenal ulcers will lead to a fatality in the absence of surgical intervention. On the other hand, it is not at all uncommon for smaller perforations to close spontaneously. I have seen an appreciable number of classical examples of perforated peptic ulcer with all of the pathognomonic signs, including gas beneath the diaphragm revealed by x-ray, a board like rigidity over the epigastrium etc., in which, upon exposing the gastro-duodenal area, it was necessary to peel away a tab of omentum and scrape off a well defined layer of plastic exudate in order to find the perforation. How to determine which perforations have sealed over or will do so, and which will remain open and produce a fatal peritonitis unless closed surgically, poses a question too delicate to quibble over. In other than an exceptional case now and

then, when, for well advised reasons, the surgeon may be reasonably certain the perforation has sealed itself off, surgical intervention is of course eminently justified—indeed imperative.

I believe surgical intervention for the removal from the stomach or intestines of any foreign body that has been swallowed by an individual—and I have particularly in mind open safety pins swallowed by children—is generally unnecessary. It has been my experience that most foreign bodies—and open safety pins have been *no* exception—will pass through the intestine with surprising ease and promptness. Hair swallowed by the insane is an exception.

I believe perforations of the intestine as a result of water blast (a condition to which Navy doctors—particularly those who served in the Pacific during World War II—are no strangers) are due to sudden compression of the submerged part and the physical principal of brissance and never to water being forced into the bowel through the victim's rectum.

I believe that by and large technically the poorest surgery which has for long been done has been rectal surgery. I believe any surgeon who is unwilling or unable to attend personally to the post operative dressings of rectal cases—especially the post operative care of fistulas—shouldn't do rectal surgery. Closely allied with rectal surgery have been the various surgical maneuvers designed for the removal of pilonidal cysts. I believe the surgical removal of a pilonidal cyst that has never been acutely involved but simply because it is there is ill advised. In the armed services and specifically at the Naval Academy, more sick days have been compiled as a result of failure to obtain first intention healing following excision of pilonidal cysts than from any other surgical procedure. This is no preeminently preferable type of operation for removal of a pilonidal cyst. All seasoned surgeons know that the multiplicity of methods recommended is in itself an attestation that no known method is ideal.

I believe that the gas responsible for gaseous distention of the abdomen in the presence of intestinal obstruction is generated within the viscus and is not to any significant extent accountable for in terms of swallowed air.

I believe that the failure of kidney function following crushing injuries is no different from the failure of kidney function under any shock producing set of circumstances. I believe the attempt to ascribe kidney failure to some element peculiar to crushing injuries in a large series of cases reported upon in London incident to the bombing raids early in World War II was in a large measure a misfire. There may be such a thing as the blockage of kidney tubules by hematin crystals or some other substance liberated by crushed muscle tissue, as the experts have tried to establish. I nevertheless believe that the kidney's tolerance of a reduced oxygen supply is very delicate and that when, by reason of a marked drop in blood pressure, which is both a cardinal cause and an effect of shock (whatever the circumstances under which it occurs, whether from a crushing wound, a gunshot wound or indeed as a result of surgery in the operating room) the oxygen supply to the kidney is reduced beyond a critical level and remains below that level beyond a critical time interval the kidney will cease to function. And whenever such a set of circumstances is responsible for that cessation it can never again be made to resume its function any more than a brain that has been deprived of oxygen beyond a critical time can be made to recover its function. I believe that kidney failure associated with injury or operation can, in the main, be explained in terms that simple, and an attempt to formulate an explanation involving some specific substance or bizarre set of principles is pointless.

While I am confident that never has so excellent a brand of medicine or surgery been available to the citizenry of this nation as at present, I believe all is far from well in the house of medicine at this time. For those who are interested in the implications of this comment—and all members of the profession of medicine should be

interested—I would invite their attention to a report entitled, "Physicians for a growing America," submitted last October by a Consultant Group on Medical Education appointed by the Surgeon General of the U. S. Public Health Service. Many of the facts brought to the fore in that report are astounding and certainly should be the concern not only of the standard bearers of medicine but of the public at large.

More definitively than the broad recommendations of the consultant group in point, I believe the following concepts should be immediately implemented:

(1) Streamline the formal course of medical education by eliminating non-essentials during the first half and point towards the specialization of the student's choice during the second half of the standard four year course in medical school.

(2) Establish a more liberal policy with respect to academic grades as a requisite for admission to medical schools. It is the aptness of a student after, rather than before, he enters upon the study of medicine that determines his competence as a doctor. Some individuals like plants bloom and bear earlier than others.

(3) Insure the availability of an adequate number of physicians to meet the needs of the armed services by the provision of an independent system of supply. The source of supply, i.e. the nation's medical schools, should be the same for both civilian and service needs. The capacities of the already existing medical schools should, through government subsidy, be increased and new schools established to meet these needs. This, I believe, should have been done a decade ago when it became obvious that the supply of physicians in America was inadequate and the likelihood of appreciable improvement by any means short of government control appeared dismal indeed.

(4) I have for long believed that military medicine was sufficiently unique in its requirements, sufficiently distinct in its mission and sufficiently meritorious through its achievements to warrant being considered a specialty sui generis, and being accorded such recognition as would be inherent in its own specialty board. This concept, I believe, has never been more bona fide than today.

(5) Bring the cost of medical care within ready reach of all of the people all of the time.

(6) Give medical research the fairest wind and the most careful and able navigation it is possible to provide. The wind and the waves are always on the side of the ablest navigators.

That the measure of the world is man has for long been recognized as a truism—but a truism in only a limited sense, to be sure. By a similar token the measure of the universe may also be said to be man—in-so-far as man personally, is able or will ever become able, to explore it. That the answer to the somewhat trite question: "Where do we go from here?" lies and will continue to lie within the province of medicine can scarcely be denied. How far and how fast man will be able to travel into space and return to earth intact must depend upon the capabilities of astronautical medical research to come to terms with the forces of nature and extraterrestrial environment. This I believe currently constitutes medicine's most formidable, but not necessarily its most important challenge.

Of greater immediate importance than flying into space, I believe, is a recognition of the demands of the times here at home—the demands of what we have to admit is progress and it devolves upon the leadership of what we are accustomed to regard as organized medicine in America to come to terms with these demands—to get into step with history that is in the making for there is only one alternative and that is to be run over by it.

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EDITOR'S NOTE: Dr. Pugh entered the Medical Corps of the U. S. Navy upon graduation from the University of Virginia in 1923 and recently retired after serving over 33 years. From 1951-1955 he was the Surgeon General in the Navy.

Plant Dermatitis

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Although plant dermatitis is frequently seen, it continues to be difficult to prevent and troublesome to treat. This paper discusses the offending plants, the treatment of the dermatitis, and the prevention of the disease.

IN FOLLOWING THE LITERATURE I have concluded that of all the sources of allergic contact dermatitis the plant kingdom provides most of the causes. In this paper some of the most common allergens, management of plant dermatitis, and hyposensitization are discussed. For practical purposes, the allergens producing dermatitis venenata may be divided into three principal groups: poison ivy and other members of the family Anacardiaceae, a heterogeneous group of plants, and pollens.

Poison ivy is the most important single cause of contact dermatitis. Because of the relative ease with which people can become sensitized to it, it has become one of the principal means for clinical and experimental studies on the subject. The recent synthesis of various components of urushiol has led to more intensive investigations in immunology and hyposensitization with the catechol derivatives. Shelmire,[1] in the 1940's, conducted extensive studies with poison ivy and weed oleoresins, and more recently Kligman[2] worked with 3-pentadecylcatechol and compared it with the oleoresins. Many others have worked extensively in the chemistry and clinical investigation of plant allergens but I do not think it necessary to mention them here.

* Adapted from a paper presented at the Thirtieth Annual McGuire Lecture Series and Symposium on Dermatology, Medical College of Virginia, November 12-14, 1958.

From the Department of Dermatology, The Cleveland Clinic Foundation, and The Frank E. Bunts Educational Institute, Cleveland, Ohio.

FAMILY ANACARDIACEAE

I thought it would be of interest to present some of the members of the family Anacardiaceae which are sources of dermatitis (Table 1). The various catechols with saturated and unsaturated fatty acid side chains are the allergens in these plants. Cross-sensitivity to the various catechol allergens is regularly found in the sensitive individual. Three-pentadecylcatechol, which I will hereafter speak of as "PDC," is the most stable of the allergens and is the one Kligman[2] used in his extensive studies.

The two plants that are the chief sources of Rhus dermatitis in the United States are poison ivy and poison oak. Poison oak is limited to the western United States from New Mexico to British Columbia. It grows only as a tall bush; its leaves are hairy on both surfaces and, although variable in shape, resemble oak leaves. Poison ivy may grow as a short bush or shrub, a vine, a trailer, or a woody vine. It is the well-known three-leaflet plant with white berries. At a certain stage the leaf tips are red. The upper surface is smooth and the under surface is hairy. Poison sumac, although of less importance, is a tall bush with from three to seven leaflets on each long, reddish stem.

As to the mode of contact, some individuals believe themselves to be so sensitive that merely being close to the poison ivy plant will precipitate dermatitis. However, it has been shown that the pollen does not contain the allergen and that even direct contact with the unbroken leaf does not produce dermatitis. Smoke that is absolutely free of unburned particles of the sap does not cause dermatitis. Because spring brings many people to the woods, and fall lures the hunter, the incidence of dermatitis is greater in these seasons than in the others. Fomites such as clothing, animals, hunting and fishing equipment, golf clubs, automobile tires, and construction machinery may carry the dried sap. For example, several attacks of dermatitis on the lower portions of the legs of a golfing friend of mine were traced to golf club heads. He knew poison ivy, but was a golfer who spent some of his time swinging clubs in the brush. He also had a habit of gently stroking his legs with the club heads.

I think we are familiar with the morphology of the dermatitis produced by direct contact with outdoor plants: the lesions tend to be arranged in streaks on the exposed surfaces, and in patches on other parts, due to the transfer of sap by the hands. The severity of the eruption and of the secondary id eruption is determined by the degree of sensitivity of the individual. The oral mucous membranes may become sensitized either by chewing the leaf or on contact with the allergens.

Figure 1 shows ivy dermatitis on the legs of a

Fig. 1. Ivy dermatitis on the legs of a woman, showing the characteristic streaked pattern of the lesions.

nurse who recently immigrated to the United States from Germany. She spent her first vacation in Florida, where she acquired her first attack. The dermatitis appeared exactly seven days after her exposure to poison ivy. The characteristic streaked pattern of the dermatitis is clearly evident.

Figure 2 shows ivy mucositis in a lad of 15 years, who, on several occasions proudly demonstrated to friends his immunity to poison ivy by chewing some leaves. He chewed them once too often! and mucositis developed, involving the mouth and tongue. The reaction appeared on the day after the exposure. Dermatitis of the skin did not occur, although a patch test with ivy oleoresin was positive.

In addition to producing the skin eruption, fever, and other toxic manifestations, poison ivy may produce other less well known manifestations, especially in severe cases. Leukocytosis and eosinophilia of the blood are common. Eosinophilia also frequently occurs during hyposensitization therapy. Eosinophilia in the tissues at the sites of lesions is common. Renal damage approaching glomerulonephritis, with fatality, as an allergic reaction of the kidneys has

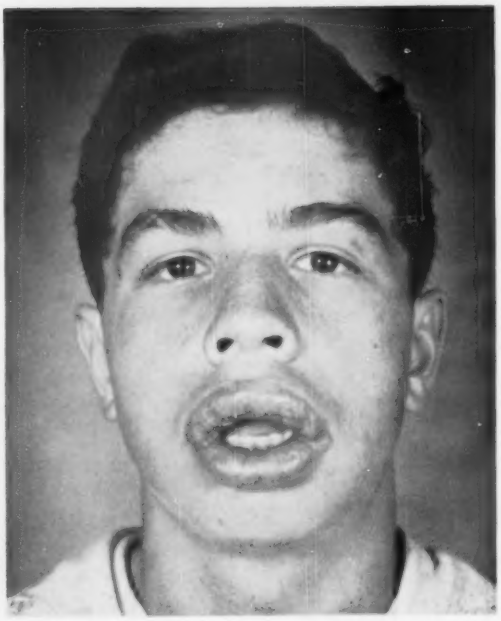

Fig. 2. Ivy mucositis on the face of a 15-year-old boy who had chewed poison ivy leaves to demonstrate his "immunity".

been reported,[4,5] although the allergy has been questioned by Templeton, Lunsford, and Allington[3] and Kligman.[2] Proteinuria has been reported[6] to occur in as high as 50 per cent of poison ivy patients. Again, these findings have not been confirmed.[2,3] Urticaria is uncommon in untreated Rhus dermatitis but common during hyposensitization. In untreated cases usually the eczematous lesions are so preponderant as to mask the anaphylactic component. Other reactions are id eruptions and dyshidrosis, which are likely to be overlooked, and occasionally there is depigmentation at the sites of the lesions.

A HETEROGENEOUS GROUP OF PLANTS

The incidence of dermatitis caused by a large number of heterogeneous plants is by no means small. Probably the greatest number of cases are contracted in an industrial environment. Edible vegetables and fruits cause dermatitis among farmers and workers on truck and fruit farms, in the processing industries, in stores, restaurants and in homes. Flowers, trees, and shrubbery cause dermatitis among workers in the horticultural and processing industries, extending through the florists and into the home. Dermatitis caused by trees, weeds, and shrubs occurs: among foresters and field workers for construction of rights of way through wooded territory; in the lumber

industry, among lumbermen, workers in lumber processing, fabrication, and chemical extraction, carpenters and persons working at home (using objects with wooden handles). Shelmire[1] routinely tests out-

TABLE 1

PLANTS OF THE FAMILY ANACARDIACEAE WHICH CAUSE RHUS DERMATITIS

Scientific name	Common name
Semicarpus anacardium	India ink tree or marking nut for laundry
Mangifera indica	Mango fruit
Anacardium occidentale	Cashew nut
Rhus toxicodendron verniciflua	Lacquer tree
Rhus toxicodendron radicans	Poison ivy
Rhus toxicodendron diversiloba	Poison oak
Rhus toxicodendron vernix	Poison sumac

door workers with extracts of 56 weeds and grasses. Most of the patients we see at the Cleveland Clinic are farmers, horticulturalists, housewives, gardeners, florists, carpenters, patternmakers, and a few are field workers. The patients cooperate by collecting samples of contact materials such as fruits, vegetables, flowers, weeds, sawdust, scrapings from wooden handles, and musical instruments, for patch tests. In addition to the extracts we have a supply of various pollens and oleoresin extracts on hand for patch tests.*

Figure 3 shows acute dermatitis in a florist, pro-

Fig. 3. Acute dermatitis on the hands of a florist, produced by contact with chrysanthemum plants.

duced by chrysanthemum. During the height of the dermatitis on the hands he experienced a generalized id eruption on the face, the V area of the chest, and

* Those who are interested in patch tests for weeds, grasses, trees, shrubs, in local geographic areas of the country, may obtain lists of plants as well as oleoresins for tests and hyposensitization, from Graham Laboratories, Dallas, Texas. We obtain pure pollens from Hollister-Stier Laboratories, Chicago, Illinois.

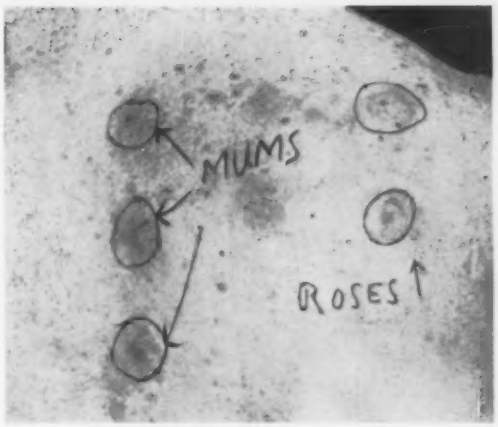

Fig. 4. Positive patch tests for chrysanthemums and rose leaves, respectively, in a garden-loving housewife.

the upper and lower limbs. Despite our attempts at hyposensitization, this patient also became sensitive to several other floral plants to such an extent that the dermatitis became nonseasonal, and he finally gave up being a florist. Figure 4 shows positive patch tests to chrysanthemum and rose leaves in a housewife who spent much time in her flower gardens.

POLLENS

Dermatitis from pollen is another type of plant dermatitis. The shell of the pollen grain contains the allergen, a resin-like material. Reactions occur mostly in persons who are farmers, gardeners, carpenters, salesmen, and field workers, who work out of doors and have contact with pollen-bearing plants. The typical seasonal appearance is often masked because of the secondary factors of chronicity, neurogenicity, and infection, which prolong the duration beyond the pollen seasons. However, in those who suffer from the dermatitis throughout the year, a careful history will show that exacerbations occur during the respective seasons for the various pollens.

The principal sites of the dermatitis are the exposed surfaces, namely, the face, neck, V area of the chest, arms, and legs. Early in the course the dermatitis is usually delineated by clothing. Figure 5 shows a patient with ragweed dermatitis. Note the sharp delineation of the dermatitis by clothing. This pattern is always suggestive of the so-called "air-borne" dermatitis. The dermatitis is usually subacute, but vesiculation may be found on the areas where pollen grains have adhered. The diffuse and even distribution of the eruption is attributed to the coalescing of minute individual papules produced by the grains of pollen.

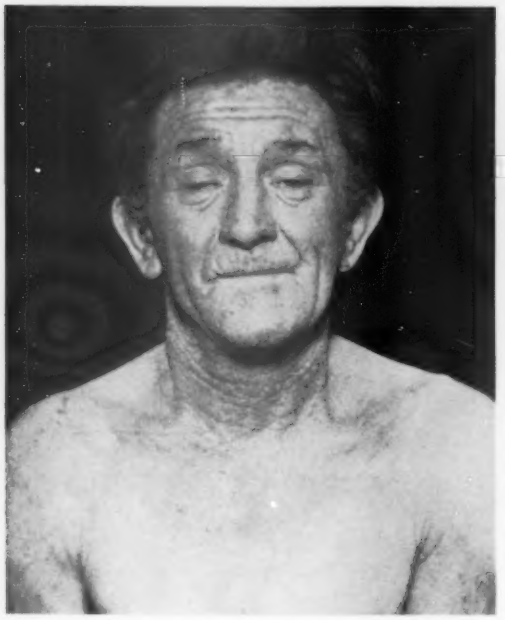

Fig. 5. Ragweed dermatitis in a man; note sharp delineation by clothing

The relationship between contact and atopic types of pollen dermatitis is of interest. In some patients, exacerbations of atopic dermatitis in the summer, aside from those due to humidity and sweating, are thought to be due to pollens. The contact dermatitis is due to the oil fraction, while the atopic or anaphylactic dermatitis is due to the water-soluble fraction. Waldbott[7] has noted exacerbations of atopic dermatitis produced by contact with pollens, and also that in atopic dermatitis, treatment with oil extracts has been effective and that in contact dermatitis treatment with aqueous extracts has been effective. We have frequently noted both the immediate and the delayed 48-hour papular response with aqueous extracts in atopic dermatitis. Occasionally, using pollen and its aqueous extract, we have obtained a positive patch test with the pollen, as well as the delayed papular reaction to the extract, in the same patient who has atopic dermatitis. This conforms to the concept that in plant sensitivity the sensitivity is systemic with both anaphylactic and eczematous antibodies.

A good history of the approximate dates of flare-ups will help to eliminate the application of an unnecessary number of patch tests. Lists of plants with the dates of pollination are a great help in selecting the most likely tree, grass, and weed pollens or oleoresins for testing. Patch tests may be applied with pollens, oil extracts, and leaves of plants.

The patient shown in Figure 6, a farmer's daughter, gave positive patch tests to pollens of ragweed, corn, and several of the grasses.

Fig. 6. Patch tests positive to pollens of ragweed, corn, and several grasses, on the back of a farmer's daughter.

MANAGEMENT

For practical purposes, it seems preferable to consider the management of plant dermatitis from three aspects: (1) topical prevention, (2) topical and systemic treatment, and (3) specific hyposensitization.

(1) Topical preventive measures include washing the skin after exposure, use of topical detoxicants, and teaching the individual to avoid the noxious plants and to be aware of possible fomites at all times. My experience with the detoxicants is limited to patients who report using them without benefit. Kligman[2] believes that detoxicants, including the ion-exchange resins, chelating agents, and the corticosteroids, are of no value in preventing dermatitis. I have learned about the inadequacy of washing the skin, from patients who have tried strong soaps, kerosene, gasoline, paint solvents, and turpentine.

(2) In topical treatment various preparations containing the corticosteroids have been ineffective in the acute vesicular and bullous stages. Antieczematous lotions such as calamine lotion or liniment containing antipruritics, wet dressings and/or baths with potassium permanganate are the most satisfactory. In subacute and chronic phases, lotions and ointments containing the corticosteroids are beneficial. Systemic treatment with corticosteroids and/or corticotropin is mandatory in severe cases, and for emotionally hyperreactive patients. The dosage equivalent of from 200 to 300 mg. of cortisone, and/or 40 units of corticotropin, daily, is indicated

during the first two or three days. Intravenous infusions of corticosteroids or corticotropin may be necessary. For some time now, the phylactic treatment with the extracts has been considered unnecessary and dangerous. Certainly the advent of corticosteroids and corticotropin has made this type of treatment obsolete.

(3) Hyposensitization in poison ivy and weed dermatitis has been extensively investigated by Shelmire[1] and Kligman.[2] Shelmire's[1] schedule with oleoresins has become well established. Kligman[2] has established a regimen with PDC. Both deplore parenteral hyposensitization with the oleoresins and PDC.

TABLE 2
ORAL HYPOSENSITIZATION WITH OLEORESINS*‡ OF POISON IVY AND WEEDS
(after Shelmire[1])

Dilution	Dose
1:100	1st week, 1 drop daily in No. 1 capsule 2nd week, 2 drops daily Thereafter increase dose more rapidly to tolerance
1:50	One half of number of drops in last dose of 1:100 dilution Increase to tolerance
1:25	One half of number of drops in last dose of 1:50 dilution Increase to tolerance

* Total dose—30 ml. each of 1:100, 1:50, and 1:25 dilutions.
‡ Time—8 to 9 mo.

Table 2 shows Shelmire's[1] method for both poison ivy and weed hyposensitization with oleoresin given orally. When the daily dose is increased by one drop weekly it usually requires eight months or more to take the required amount. In mild to moderate sensitivity, hyposensitization may require only two or three months. The regimen should be repeated every year.

The schedule of oral hyposensitization with PDC devised by Kligman[2] is shown in Table 3. He states

TABLE 3
ORAL HYPOSENSITIZATION WITH PDC
(after Kligman[2])

Week	PDC 10% in ethyl alcohol with 2.5% Aerosol OT and 0.1% Tenox Dose
1	1 drop daily
2	2 drops daily
3	3 drops daily for 4 days; 4 drops daily for next 4 days
4 to ?	Increase by 1 drop every 4 days to daily total of 30 drops until a total dose of 35 ml. has been taken.
Maintenance	5 drops or less daily

that PDC may not be available in the immediate future because of the costliness of synthesis. The drops are added to a full glass of water, stirred with a glass rod, and drunk through a disposable straw. For the average patient the dose of 30 drops daily is continued until 35 ml. has been ingested. A daily dose of 5 drops or less will usually maintain hyposensitization. When PDC becomes readily available it will probably be dispensed in tablets, because of its stability.

Kligman's[2] method of parenteral hyposensitization is shown in Table 4. He believes that this schedule

TABLE 4
PARENTERAL HYPOSENSITIZATION WITH PDC
(after Kligman[2])

Week	PDC 10% in sesame oil with 0.1% Tenox for intramuscular hyposensitization Solution (ml.)	PDC (mg.)
1	0.03	3
2	0.60	6
3	0.10	10
4	0.20	20
5	0.40	40
6	0.60	60
7	0.80	80
8	1.00	100
9-31	1.00	100
Maintenance: every 6 to 8 weeks	1.00	100

of treatment by intramuscular injection of PDC is conservative and provides adequate hyposensitization. However, the method is impractical: too many injections are required and adverse reactions are too numerous and severe. Oral hyposensitization is preferable.

Untoward reactions in oral hyposensitization are urticaria and other id eruptions, stomatitis, pruritus ani, and less often flare-ups of healed sites, and dyshidrosis. According to Kligman[2] severe reactions practically never occur in conservative oral hyposensitization. In some patients, continuation of hyposensitization is blocked by side reactions. Small doses of corticosteroids or of corticotropin, 40 to 80 units twice weekly, will suppress the reactions so that treatment can be continued.

There has been much discussion relative to the value of quantitative patch testing during the hyposensitization regimen and during maintenance of the hyposensitivity. While the method of control seems ideal, in office practice it is impractical. The end point for determining the progress of hyposensitization is the highest dilution that produces a positive

reaction at each testing. Interpretation of the results involves evaluation of reduction in degree of the dermatitis and the increase in concentration necessary to produce dermatitis by the patch test.

The advantages of hyposensitization are threefold: (1) shorter duration of an attack of dermatitis, (2) less spread of the eruption, and (3) milder dermatitis. The protection is partial and relative, depending on the degree of exposure. Estimates of the duration of hyposensitivity vary from months to years. In controlled experiments among prison inmates, Kligman[2] found that maximum hyposensitization lasted for from four to six weeks after oral prophylaxis and for from eight to 10 weeks after intramuscular treatment, after which the hyposensitivity began to wane slowly until maximum sensitivity returned in about six to eight months.

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Hospitalization of Child

The emotional shock that may occur when a child is hospitalized can be overcome when the mother stays with him, according to a New Haven, Conn., pediatrician. Writing in the February Journal of Diseases of Children published by the American Medical Association, Dr. Albert J. Solnit of the Child Study Center and department of pediatrics, Yale University, said "hospitalization of the child with the mother has served to overcome both physical and psychological difficulties.

"For young children and their parents, the hospital environment has represented a psychological hazard. The children are threatened by separation from their parents, fears of abandonment, and painful, frightening procedures in the hands of strangers.

"Since the mother may have the best access to the distorted fears of the young child, she can most effectively reassure her child. . . ."

Therefore, the mother is not replaced by the nursing or medical staff. On the contrary, the physician and nurse help the mother take care of her child.

The Educational Work of Robley Dunglison, M.D.

At The University of Virginia

CHALMERS L. GEMMILL, M.D.
Charlottesville, Virginia

Dr. Robley Dunglison was born in Keswick, England, in 1798. He came to the University of Virginia in 1825 and taught anatomy, surgery, the history of the progress and theories of medicine, physiology, materia medica and pharmacy. His salary was fifteen hundred dollars with an additional amount from student fees.

IT WAS on January 25, 1819, that the General Assembly of Virginia passed the enactment that the University of Virginia was to be established in the County of Albemarle. However, it was not until 1825 that the University of Virginia was opened with twenty students enrolled in the School of Anatomy and Medicine. Between 1819 and 1825, Mr. Jefferson was very active in establishing a definite policy for the medical school. In his "Report to the Commissioner" he stated that since there were no hospital facilities for the University, it was proposed that a single professor for both medicine and anatomy be selected. This professor would lecture on the history of medical and anatomical theories from Hippocrates to the present. Vegetable pharmacy would be taught in the botanical course and mineral and chemical pharmacy in the courses of microbiology and chemistry respectively. This instruction was to be a foundation on which the student was to build his clinical knowledge which was to be gained elsewhere. The Board of Visitors on April 7, 1824, accepted Mr. Jefferson's ideas and stated that "In the School of Anatomy and Medicine shall be taught anatomy, surgery, the history of the progress and theories of medicine, physiology, pathology, materia medica and pharmacy" and it was decided to obtain a single professor to teach all of these subjects.

It was now the job to obtain a suitable man to fill this chair. After surveying possible men in this country and finding none suitable, the Board of Visitors in 1824 sent Francis Walker Gilmer to England in order to find a man abroad. It is of interest to note that the position was to be a full-time position with no income from private practice. Mr. Gilmer was able to persuade Robley Dunglison to take the position.

What were the qualifications of Dr. Dunglison? He was born in Keswick, England, in 1798. After a classical education, he commenced his study of medicine as an apprentice to John Edmondson in his home town. After one year with Dr. Edmondson, he went to London as an assistant to Dr. Haden. He then went to Edinburgh in order to attend a course of lectures and later attended a series of lectures in Paris. He thus was under the most brilliant group of medical professors of his day. In 1818 he passed his examinations at the Royal College of Surgeons and the Society of Apothecaries in London which gave him the right to practice medicine. Later he obtained an M.D. degree at the University of Erlangen in Germany, by examination and the submission of a thesis on "Neuralgia". At an early age, he started to write and continued to write throughout his life. In fact, it was his writings that attracted Gilmer to him in London and lead him to offer the position here.

In the collection of letters of Dunglison in our library there is one from George Birbeck recommending Dr. Dunglison to Mr. Gilmer, dated 29th of August, 1824. It states "This gentleman in consequence of habits strictly literary and a taste leading to actual investigation, with a decided tendency to

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prefer the contemplative species of occupation to the more bustling, would be well placed in the University of Virginia, and I am persuaded that he would there not only distinguish himself but greatly benefit the students who might be placed under his direction." The next letter is from Dr. Dunglison to Mr. Gilmer, dated September 5, 1824, accepting the chairs of Anatomy and Chemistry. The contract between Mr. Gilmer and Dr. Dunglison stated that he would teach, with proper diligence and to the best of his ability, anatomy, surgery, the history of the progress and theories of medicine, physiology, materia medica and pharmacy and for this, he would be paid fifteen hundred dollars with an additional amount from student fees. He would also be entitled to occupy one of the pavilions of the University free of rent.

The collection of student fees bothered Dunglison. In a letter dated April 3, 1826, to the Board of Visitors, he states that the requirements for Latin should be modified for the medical students. He thought that this requirement was retarding the enrollment in the medical school, for his salary was less than he had reason to expect.

At the time of the offer, Dr. Dunglison wished to get married and this salary offered him sufficient security. He accepted the post and arrived in Charlottesville in 1825. Mr. Jefferson was very pleased with Dr. Dunglison. This was remarkable for, as Dr. Dunglison stated later, Mr. Jefferson had little faith in doctors stating that "It is not to physic that I object so much as to physicians".

Dr. Dunglison gave his lectures in these many subjects justifying this method by stating that students would not be distracted by discordant statements of many professors and repetition would be avoided. He remained at the University for eight years, leaving for the University of Maryland in 1833 and later going to Jefferson Medical College.

In addition to his teaching duties, Dr. Dunglison continued to write while in Charlottesville. He published with George Long, in 1829, an Introduction to the Study of Grecian and Roman Geography and in 1832 published his "Human Physiology". This text went through seven editions, the last one in 1856. In 1833, he published his "New Dictionary of Medical Science and Literature". This dictionary reached its 23rd edition in 1897 and sold 55,000 copies. His physiology and dictionary were his chief contributions during his professorship in Charlottesville. Also, he was one of the founders and editors of the Virginia Literary Museum and Journal of Belles-Lettres, Arts and Sciences, a weekly periodical, issued at Charlottesville, in the interest of the University. Dr. Dunglison wrote many of the articles in this journal on numerous subjects such as Modern Improved System of Road-Making, Penitentiary Discipline and Sanscrit Language.

As a scientist he aided Beaumont in his famous experiments on the gastric juice of Alexis St. Martin. Beaumont, in his book on "Experiments and Observations on the Gastric Juice and Physiology of Digestion", states that he sent a sample of gastric juice to Professor Dunglison who with the assistance of the Professor of Chemistry of the Virginia University made an analysis. Beaumont in his book, gives in part a letter from Dr. Dunglison dated February 6th, 1833. In this letter, Dr. Dunglison described that free muriatic (hydrochloric) acid was found in the gastric contents. Beaumont states "Those who are acquainted with him, know his candor and fairness are above the reach of suspicion."

There have been many tributes to Dr. Dunglison. Dr. Radbill, in a recent article on Dunglison, states that Dunglison was a teacher in all he did. He was a friend to the young, his writings were widely used by students and doctors. His home was noted for its sociability. He favored preliminary education in Mathematics, Languages, English and Sciences. He introduced into the curriculum the teaching of the History of Medicine. It is of interest that he was the first full-time professor of medicine in this country with his salary coming from the University and students and not from private practice. It was under his guidance that the School of Medicine of the University of Virginia was started.

Osler states that Thomas Jefferson did a good work when he imported him from London, as Dunglison had all the wisdom of his day and generation combined with a colossal industry. Gross, in "A Memoir of Robley Dunglison", says that Dr. Dunglison was no ordinary man; indeed, in more than one sense of the term, he was an illustrious man, a great scholar, an accomplished teacher, a profound physiologist, an acute thinker, a facile writer, a lucid, erudite and abundant author.

In conclusion, I would like to quote from Sarton, "Too many of our scientists are technicians and nothing more. Our aim is to humanize science and the best way of doing that is to tell and discuss the history of science. If we succeed, men of science will cease to be mere technicians and will become educated men."

The author wishes to thank Miss Elizabeth Atkins, Dr. William Bean, Dr. Samuel X. Radbill and Mr. William H. Runge for their aid in the preparation of this paper.

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New Psychotherapeutic Drug

A new psychotherapeutic drug, methaminodiazepoxide (Librium) has produced "impressive" results in the treatment of various mental disorders, according to Dr. Titus H. Harris, a Galveston, Texas, psychiatrist.

In a preliminary communication in the March 12th Journal of the American Medical Association, Dr. Harris said the drug reduces anxiety and agitation but does not cloud consciousness or impair intellectual function.

"My experience with its use in all types of anxiety, such as convulsive disorders, tension states, obsessive-compulsive conditions, agitated depressions, and jitters in alcoholics indicated that methodaminodiazepoxide is one of the most interesting drugs of its type that has been developed."

"Of special interest is its apparent effectiveness in obsessive-compulsive neuroses, which have thus far defined chemotherapeutic management."

Dr. Harris said the drug exerts three primary actions—tranquilization, muscular relaxation, and anticonvulsant effect.

"At a recent symposium . . . investigators in a number of medical specialties, including internal medicine, cited impressive results obtained from use of methaminodiazepoxide in a variety of conditions and a freedom from side-effects at therapeutic dosage levels. These investigations confirm my findings. . . "

The drug was described as "one of a new class of compounds" by Dr. Harris, chairman and professor of the department of psychiatry and neurology, University of Texas Medical Branch. He said its unique action on vicious, agitated monkeys, dingo dogs, and other wild animals prompted the first clinical trials.

The Control of Weight in Hypertensives on Rauwolfia and its Derivatives

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In the obese hypertensive patient there is need of weight control as well as reduction of blood pressure. Rauwolfia is a useful hypotensive drug but may stimulate the appetite and cause a gain in weight. Many of the appetite suppressing drugs raise the blood pressure. A new drug appears to be a solution to this dilemma.

IT IS WELL KNOWN that obesity is particularly dangerous to the hypertensive. Indeed it is even possible that obesity may be an etiological factor in the development of hypertension for not only is it often noted in conjunction with hypertension, but obesity may in many cases precede elevation of blood pressure. Furthermore, it has been amply confirmed that weight reduction in many cases will lead to a reduction of blood pressure and that maintenance of a desirable weight may be accompanied by maintenance of normotensive levels of blood pressure. Therefore, it behooves the physician who is dealing with the obese individual and particularly with the obese hypertensive to encourage weight reduction and help the patient to achieve it. This in fact should be the first step in a regimen of therapy for moderate and mild hypertensives who are obese, for often they will need no further therapy. If, however, the patient's hypertensive state is of such a degree to require more than simply weight reduction, this can now be achieved by the use of a variety of relatively recently developed pharmacological agents that have potent and anti-hypertensive effects. Perhaps the most important and widely used among these agents have been rauwolfia and its derivatives alone or in combination with other anti-hypertensive drugs. However, in the obese hypertensive, rauwolfia has proved to be something of a "two-edged sword" for there is now suitable evidence to indicate that rauwolfia may stimulate the appetite while it is bringing down the blood pressure and this may often be accompanied by excessive gains in weight. The undesirability of this side effect of rauwolfia is obvious and often this secondary effect of the rauwolfia derivatives have more than off-set the benefits gained from its anti-hypertensive action. Indeed the appetite stimulation that occurs with rauwolfia preparations has been sufficiently pronounced so as to have lead to their use as an appetite stimulant in patients who are undernourished and in children who are eating poorly. The practicing physician therefore is faced with something of a quandary in dealing with the obese hypertensive patient who requires mild drug therapy and in whom rauwolfia derivatives seem to have an appropriate place in management. On one hand the physician is impelled to use a drug which he knows will help the patient's hypertensive condition and on the other hand he must be aware of an undesirable appetite stimulant, which will intensify the desire to overeat and hamper attempts to have the patient reduce weight concomitant with the anti-hypertensive treatment. It is hard indeed to get the average obese individual to adhere to a program of weight reduction. It is considerably harder to achieve this goal in a patient whose appetite is unduly stimulated by a pharmacological agent.

The solution to this dilemma would appear to be the administration of yet another agent to suppress the appetite and thereby counteract the undesirable side effect of the rauwolfia preparation. The need to utilize means of helping the patient curtail his caloric intake is clear. Unfortunately the commonly used appetite suppressant drugs are unsuitable for use in the hypertensive subject, because these drugs tend to overstimulate the patient and often result in a further elevation of blood pressure.

The recent development of a new anorexigenic drug has offered an opportunity to resolve this problem. Experience with levo-1-phenyl-2-aminopropane alginate (LEVONOR) has indicated that it is a

potent anorectic drug that is remarkably well tolerated and is not accompanied by the usual analeptic effects. In clinical trials with this preparation, patients have been found to achieve appetite suppression without either stimulation or blood pressure elevation.

The study reported here was undertaken to determine the effectiveness of this new anorexigenic medication in preventing undesired weight gains during rauwolfia therapy of obese hypertensive subjects.

METHODS AND MATERIALS

Forty-one private patients who were being treated for hypertension were selected for study. All of these patients either were receiving or in the past had received some form of rauwolfia. The study plan included a diet that was low in salt and low in calories, and each patient was thoroughly schooled and educated with respect to dietary management. In addition, the patients were given a derivative of rauwolfia in order to obtain satisfactory blood pressure levels, and as an adjunct to therapy each was instructed to take levo-1-phenyl-2-aminopropane alginate (LEVONOR)—a dosage of 5 mg. three to four times a day, the last dose being given at 8 p.m. —a few patients were given even larger doses. The treatment was continued for periods of five to forty-four weeks.

As the study proceeded it was clear that the patients could be classified into two groups. Group One included individuals who appeared generally amenable to medical regimentation and followed their diet and pharmacological therapy rigidly. Group Two included those patients who were less cooperative and less reliable in following therapeutic regimen. Group One comprised fourteen male patients and sixteen females ranging in age from 28 to 58 years. Before treatment, sixteen of these patients weighed in excess of 200 lbs., and five in excess of 240 lbs.—one weighed 290 lbs. The average pre-treatment weight for this group was 204.5 lbs. Group Two included one male and ten females ranging in age from 34 to 68 years. Six of these patients weighed in excess of 200 lbs.—three weighed 214 lbs. The lightest patient in this group weighed 168 lbs. and the average weight of patients prior to treatment was 179 lbs. This group of patients in whom less favorable results could be expected generally were found to have their hypertensive disease more firmly established and at somewhat higher levels. In addition, several of these patients suffered concomitantly from one or more other conditions, including chronic cystitis in two females, diabetes and proctitis in one, and duodenal ulcer, arthritis and infectious hepatitis, acne, edema and menopausal complaints in others. As a group, these patients appeared to exhibit marked emotional disturbances and anxiety from domestic problems.

RESULTS

Blood pressure levels were controlled by the use of rauwolfia or one of its derivates in each patient studied in this series. Every patient in Group One lost weight during the course of the study, with individual losses ranging from 5.5 to 34 lbs. Loss of weight of 2 lbs. a week or more were noted in seven patients, and fifteen more had reduced their weight on an average of 1 to 2 lbs. a week. Eight patients showed less than one pound a week weight loss. The average over-all weight loss of patients in this study group was approximately 16 lbs.

The patients in Group Two had less satisfactory results. Ten patients succeeded in losing weight while one gained 7½ lbs. This patient, a 34 year old woman, weighed 188½ lbs. at the start of treatment and reached 196 lbs. before being dropped from the study as completely uncooperative. The over-all average weight loss of the eleven patients in this group was 0.33 lbs. There was, however, considerable variation in weight loss exhibited by these patients. While some were very careless about taking medication, others showed varying degrees of cooperation at different period while under study. One 68 year old woman who weighed 214 lbs., and suffered concomitantly from rheumatoid arthritis was given steroid therapy for the latter disease in addition to the low salt, low calorie diet. She succeeded in losing 11 lbs. in 34 weeks despite the tendency to retain sodium and water which might be expected from her steroid therapy. One patient in this group, a 34 year old male who weighed 214 lbs. was given a placebo for a four-week period instead of the anorexigenic drug. During this period he gained 1.5 lbs. Subsequently, the placebo was substituted for 5 mg. levo-1-phenyl-2-aminopropane alginate (LEVONOR), and during the next 4 weeks he lost 7.2 lbs. or an average of 1.8 lbs. per week.

As might be anticipated, the degree of success in the weight reduction and blood pressure reduction program tended to vary directly with the degree of cooperation exhibited by the patient. By and large, those patients who were most cooperative succeeded in losing the greatest amounts of weight, and in these patients blood pressure reductions were most dra-

matic. In patients in whom cooperation was less satisfactory weight loss tended to be less marked and blood pressure control less satisfactory. Thus a 37 year old man who weighed 274 lbs. at the onset of the study lost 34 lbs. in six weeks for an average of 5.6 lbs. per week. His blood pressure fell to the upper limits of normal. Another patient, a 36 year old woman, weighing 185 lbs. lost 21 lbs. in six weeks for an average of 3.5 lbs. weekly and her blood pressure similarly fell dramatically. On the other hand, an uncooperative 36 year old woman who weighed 175 lbs. failed to take her prescribed medication and was not cooperative with respect to her dietary restrictions. She lost only 2 lbs. in twelve weeks, and the reduction of blood pressure was only slight. Another 53 year old female beset by domestic problems who weighed 214 lbs. succeeded in losing only 5 lbs. in six weeks of treatment for an average of 0.83 lbs. per week. She too had a relatively unsatisfactory blood pressure response.

DISCUSSION

The relationship between obesity and hypertension is well established. All authorities agree that obesity tends to aggravate hypertension, and in many cases precedes it in such a fashion as to suggest a possible relationship in the pathogenesis of the hypertensive state. Whether or not there is a cause or relationship between the two conditions, certain it is that the hypertensive subject is benefited by weight reduction. Many hypertensive patients, however, require the use of drug therapy in addition to dietary therapy in order to satisfactorily control blood pressure levels.

In recent years, in fact, there has been an increasing tendency to utilize pharmacological agents to reduce blood pressure in earlier and milder cases of this disorder with the view that control of blood pressure might very possibly reduce the danger of undesirable and even lethal vascular complications from the hypertension. Unfortunately, a weight reduction program based on caloric restriction alone is often unsuccessful in the management of the hypertensive obese subject, and many of these patients do far better with respect to maintenance of caloric restriction by the adjuvant use of an appetite suppressant. Most of the drugs utilized for this purpose have been the amphetamines, and these drugs tend to stimulate the central nervous system of the patient to whom it is administered, and at the same time often will lead to the further elevation of blood pressure. Similarly rauwolfia and its derivatives, one of the most useful therapeutic agents in the management of mild to moderate degrees of hypertension, are often accompanied by the undersirable side-effect of appetite stimulation so that many patients to whom this drug is administered gain weight. This situation presents the physician with something of a dilemma in the managing of the obese hypertensive. On one hand it is desirable to reduce blood pressure, but on the other it is equally desirable to reduce weight. Neither of these things can be accomplished in many patients without the use of drug therapy, and unfortunately the drug that is suitable for blood pressure reduction in many of these patients causes augmentation of appetite; whereas, the drugs that have been used to curb appetite tend to aggravate the blood pressure.

The recent development of a new appetite suppressant drug, levo-1-phenyl-2-aminopropane alginate (LEVONOR) appears to provide a solution to this uneasy dilemma. This medication, while curbing appetite, appears to have little, or any, analeptic activity. It seemed therefore that this drug would be ideally suited for use in the overall management of the hypertensive obese subject, particularly when the patient was being given rauwolfia or one of its derivatives in order to control blood pressure. This paper reports results in the series of 41 obese hypertensive patients who were treated with both rauwolfia and levo-1-phenyl-2-aminopropane alignate (LEVONOR) along with a low salt, low calorie diet. The anorexigenic drug was administered three or four times daily before meals in 5 mg. doses, and in many cases the fourth 5 mg. tablet was administered at 8 p.m. as well. These patients showed an excellent response both in terms of blood pressure and appetite suppression. In all patients who were cooperative, good weight reduction was achieved along with reduction of blood pressure to satisfactory levels. As might be expected, results were less striking in patients who were less cooperative. These patients lost less weight and exhibited less satisfactory blood pressure control. However, forty of the forty-one patients succeeded in reducing their weight to some degree while only one gained weight. None of the patients exhibited any signs of undue central nervous system stimulation despite the fact that the medication was often given as late as 8 p.m.

The findings in this study tend to confirm those of earlier studies with respect to the efficacy of levo-1-phenyl-2-aminopropane alginate (LEVONOR) as an appetite suppressant in obese individuals. Comparison of this drug with other anorexigenic preparations shows it to be fully as effective as any in suppressing appetite, and at the same time less

stimulating and better tolerated. This study appears to establish it as the drug of choice in the management of the obese hypertensive individual in whom weight reduction is particularly important and in whom appetite may be further stimulated by the use of rauwolfia preparations to control hypertension. It would appear likely that this drug would be well suited for the management of the obese diabetic who poses many similar problems in therapy and in whom unwanted weight gain, particularly with the use of oral hypoglycemic agents may be an undesired side effect.

SUMMARY AND CONCLUSIONS

Forty-one obese hypertensive patients were maintained on a low calorie, low salt diet with the administration of rauwolfia derivatives to control hypertension and levo-1-phenyl-2-aminopropane alginate (LEVONOR) as an appetite suppressant. All but one of the patients succeeded in losing weight while on this regimen and in almost all the blood pressure was satisfactorily controlled. In general, weight reduction and blood pressure control was found to be proportional to the degree of cooperation exhibited by the patient. Levo-1-phenyl-2-aminopropane alginate (LEVONOR) appears to be an ideally suited anorexigenic drug. It succeeds in curbing appetite without any untoward and undesired central nervous system stimulation or aggravation of blood pressure elevation, and at the same time allows rauwolfia and its derivatives to be administered without concomitant appetite stimulation so frequently noted with these preparations.

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Restoring Invalids to Active Life

An improved electronic pacemaker, a device that stimulates the heart, is restoring incapacitated cardiac patients to an active life. The device, originally designed for persons who had undergone heart surgery, is now being used increasingly for nonsurgical patients who sustain heart block as a complication of various afflictions of the heart muscle, according to the April 30th Journal of the American Medical Association.

The portable, lightweight, transitorized pacemaker consists of a self-contained battery of long life which delivers a repetitive electrical stimulus by means of a stainless steel electrode which is implanted in the heart and runs through the chest wall to the externally worn pacemaker. Because the entire apparatus is self-contained and only slightly larger than a package of cigarettes, the patient can use it with complete freedom of movement. For this reason it has been found to be particularly useful for persons who, after the heart block occurs, are incapacitated because of intermittent fainting spells or because their heart is too weak to permit physical activity.

The article was prepared by C. Walton Lillehei, M.D.; Vincent L. Gott, M.D.; Paul C. Hodges, Jr., M.D.; David M. Long, M.D., and Earl E. Bakken, B.E.E., department of surgery and the Variety Club Heart Hospital, University of Minnesota Medical School, Minneapolis, on the basis of 66 surgical and nonsurgical patients at the University of Minnesota Hospitals who used the miniature pacemaker.

The longest continuous stimulation of a nonsurgical patient's heart by means of the device has been 15 months, and it was still in use. "The question of how long stimulation can be maintained appears to be related to electrode materials, design, and technique of implantation. If an electrode does fail, another may be implanted."

The authors also said a future refinement of such devices probably will be the use of electromagnetic induction to replace the electrode.

". . . experimental work in dogs has indicated the feasibility of implantation of a tiny coil within the myocardium and the use of electro-magnetic waves through the intact chest wall from a transmitter on the skin surface."

Portal Caval Shunt

A Reappraisal

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Patients, who in the past would have been doomed to progressive liver disease and its complications, are now being salvaged by shunt operations on the portal vein. A better understanding of the dynamics of the portal circulation in cirrhosis of the liver has been necessary in developing these procedures.

THE STATUS of a portal caval shunt procedure has changed considerably in recent years and within the past few months. In the past, the portal caval shunt or spleno-renal shunt was primarily reserved for those patients who had multiple episodes of esophageal bleeding for varicosities or the occasional patient with a Banti-like syndrome consisting of hypersplenism and the coexistence of esophageal varices. Indeed, the presence of ascites was thought by many to contraindicate a shunt procedure and this concept has been held by many until recent investigative procedures have shown this to be incorrect.

To understand the formation of ascites in portal hypertension one has only to understand the various stages of decompensations which occur following the cirrhosis and peri-portal scarring. Initially, the smaller venous radicals within the liver are capable of conducting the portal blood through this diseased organ. As the scar tissue increases the peri-portal obstruction, the collateral vessels within the liver parenchyma itself are unable to conduct the normal flow at a satisfactory rate through the liver bed, and a back flow or portal hypertension occurs. This may initially manifest itself only by distension of the portal vein with increase in pressure in the splenic and mesenteric vessels, edema within the mesentery of the small bowel and "sluggish" absorption of nutrients from the G.I. tract. A further complication is the inadequate transportation of the products of nutrition through the portal system, and incorrect synthesis within the liver. This produces a nutritional deficit in the patient already suffering from a chronic illness and a diseased liver. Next is the development of progressive edema of the mesentery and lymphatics in this region with a gradual weeping or outpouring of serous protein transudate through the portal bed, mesenteric and splenic vessels. This is the beginning of the accumulation of ascitic fluid which in the past has been thought to contraindicate portal caval shunt. The increase in portal backflow causes a similar increase in pressure within the esophageal, coronary, and gastric veins and the development of enlarged, distended and thin-walled esophageal vessels. Bleeding may or may not occur from these dilated varicosities; however, if bleeding does not occur, the ascitic fluid will continue to accumulate, eventually producing the typical far advanced post-cirrhotic patient with massive ascites, distended abdomen, enlarged vessels over the upper chest and abdomen (Caput Medusae) and demonstrable esophageal varices either by esophagoscopy or as demonstrated by barium swallow.

This patient in his far advanced state has a low total protein because of the continual loss of transudate high in albumin and globulin from the bloodstream into the free peritoneal cavity. A severe malnutrition occurs with significant loss in body mass although the patient's weight may remain fairly stable due to accumulation of functionless ascitic fluid. Such a patient almost certainly has no chance of recovery unless the tension in the portal system can somehow be reduced or shunted to reverse this lethal sequence. It is of course obvious that the liver itself must have enough functioning parenchyma to support life. It has been estimated that a great mass of total liver can be destroyed (90%) before reversal of hepatic function occurs. This must be true, since a massively cirrhotic liver can apparently support life if the distended venous radicals within

its system and in the portal bed are adequately decompressed.

This paper then concerns itself with viewing the cirrhotic, ascitic patient as a surgical candidate when properly evaluated by liver function studies and when seen from the viewpoint of a mechanical venous obstruction which must be improved to reverse the unrelenting and progressive portal hypertension. There is no known drug which can do this—there are most certainly, diuretics which will temporarily reduce the ascites by renal mobilization of fluid. There are methods such as multiple paracenteses which can remove the ascitic fluid but with concomitant loss of protein material and progressive wasting of the patient. There are diets low in protein to prevent ammonia intoxication and to afford a high carbohydrate and high vitamin supplement. These most certainly are adjunctive methods of treatment for the patient with severe liver disease and ascites, but they in a sense are similar to the medical treatment of a patient with mitral stenosis and failure. Digitalis and diuretics are but temporizing agents when used alone, that is, when the underlying condition of stenosis has not been surgically corrected.

sion of the portal blood through gastro-esophageal vessels and development of esophageal varices, that this not only fails to decompress the portal system but decreases the blood flow through the liver, increases the possibility of rupture of these thin-walled varicosities, and further decreases the transportation of nutrient material to the liver parenchyma. Warren and Muller[3] have performed rather conclusive studies on the hemodynamic changes in cirrhosis in which they strongly emphasize the role of the obstructive factor in the cirrhotic liver as related to the development of portal hypertension. McDermott[3] has also shown the importance of the combined hepatic and portal decompression in the treatment of ascites. This contrasts with those authors[4] who have felt that portal hypertension is contributed to by the development of arterio-venous fistulae in the gastro-intestinal tract especially in the stomach. The work of Warren has been further verified by our experience and is shown briefly in the pressure studies of the accompanying diagram. The increase of portal pressure on the hepatic side of the clamp does not support the concept of pressure elevation maintained by hepatic arterio-venous fistulae but rather by the obstructive

PORTAL VEIN PRESSURE STUDIES

	FREE PORTAL PRESSURE PRE-SHUNT	HEPATIC PORTAL PRESSURE WITH PORTAL VEIN OCCLUDED	POST SHUNT PORTAL VEIN PRESSURE
Case 1	310 mm. H20	300 mm. H20	90 mm. H20
Case 2	290 mm. H20	290 mm. H20	100 mm. H20
Case 3	300 mm. H20	280 mm. H20	120 mm. H20

RESEARCH STUDIES INDICATING THE VALUE OF PORTAL CAVAL SHUNTING PROCEDURES

The side-to-side portal caval shunt is specifically mentioned since it serves to decompress the engorged venous bed within the liver as well as to decompress the more distal portal system. The end-to-side shunt leaves the hepatic venous bed undecompressed, and as demonstrated by pressure studies proximal and distal to the clamped portal vein at the time of operation. An elevated portal pressure on the hepatic side of the clamp indicates elevated pressure within the hepatic venous system and suggests that a side-to-side shunt is necessary for hepatic decompression. Rousselot[1] has shown that the rate and the pressure of the hepatic blood flow is not changed in the presence of increasing collateralization. The development of esophago-gastric varicosities decreases the hepatic blood flow but the portal tensions are not decreased. This study concludes that with diver-

theory of back pressure due to venous occlusion in the venous channels. Pressure determinations on each side of the portal clamp and wedge pressures of the hepatic venous bed reveal that increased pressures in the proximal or hepatic system regularly occur in the cirrhotic patient. This indicates to us the value of the side-to-side shunt to decompress the hepatic as well as the general portal circulation.

The presence or absence of ascites or esophageal varices indicates that this particular stage of development in the disease process has not occurred. Certainly, portal hypertension of a significant degree will eventually cause a decompression through the collateral channels—the coronary veins—and esophageal varices will result. Accordingly, portal hypertension of a significant degree will eventually cause a decrease in motility of the small intestine with resultant edema, transudation and ascites. The full-blown ascitic patient with portal hypertension and esophageal varices has arrived at the end stage of his

disease. It seems logical that such a stage is undesirable and that earlier surgery is indicated. Welch[5] has performed an interesting clinical study on the side-to-side portal caval shunt in the treatment of ascites. In his series, fourteen patients with cirrhosis and ascites were operated upon using the side-to-side shunt technique. This author had satisfactory results with this procedure on all fourteen patients with significant reduction or disappearance of ascites, decrease in malnutrition of the patient, and in many instances return to a normal existence. Such a shunt, he points out, provides new egress from the congested liver and allows, as well, a certain amount of the portal blood to reach the liver bed. It should be mentioned that there are those who feel that side-to-side shunt is not strongly indicated. Among these have been Linton, Child, and Madden[6] (Discussion on the topic "Surgery for Ascites and Cirrhosis"—Ann. Surg., September 1959). These authors apparently feel that a side-to-side shunt not only removes the portal venous blood from the liver, but also some of the hepatic arterial blood is shunted into the inferior vena cava, thereby depriving the liver of both its venous and arterial supply. This reasoning seems to have been disproven by the clinical results of both Warren, Welch and our own series. We have had no incidents of liver failure in the three patients thus far studied, nor have Welch or Warren.

Furthermore, the portal system once decompressed allows better absorption of food from the G.I. tract and decreases protein loss which otherwise continually occurs through transudation. The patient with esophageal varices with or without a history of bleeding provides a further indication for portal caval shunting.

The technical procedure of the shunt is no longer significantly formidable. A liver which is decompensating with elevated blood ammonia levels and early ammonia intoxication certainly provides a strong contraindication for surgery. However, if the patient is alert, has liver function studies within a fair range of normal, especially has adequate serum proteins, then we consider a side-to-side portal caval shunt an indication with or without varices if ascites is present. If varices alone are present without ascites we also consider this an indication for the shunt procedure.

This operation is best done through a right thoracoabdominal approach, dislocation of the right lobe of the liver into the chest and clear identification of the portal vein and vena cava.[7] Frequently, the caudate lobe of the liver must be resected in order to bring the inferior vena cava and portal vein together without undue tension and to provide an adequate lumen for anastomosis. This procedure can be done without significant bleeding or difficulty. Pressure studies are taken at the time of operation of the free flow within the portal vein and flow on the hepatic side of the clamp. Wedge pressures of the hepatic venous bed are also measured. A side-to-side shunt is then performed making the anastomosis approximately 1½ cm. in size.

The patient facing such surgery is given antibiotics for several preoperative days and also treated with Vitamin K and transfusions as seem indicated.[8] Blakemore has emphasized the importance of a "crash" program prior to surgery on cirrhotic patients. This refers to multiple transfusions, adequate protein and vitamin diet, etc.

PRESENTATION OF CASES

Case 1—J. M.—45 year old white male. This patient had a long history of ascites requiring multiple paracenteses, hepatic function studies on repeated occasions indicated cirrhosis with alcoholic history as the basis. The patient had not worked for six months, had become progressively debilitated with a markedly distended abdomen, extreme weight loss and cachexia. Liver function studies showed a B.S.P. of 39%, a total protein of 5 gms. with a reversal of the A G ratio. Patient also had a slightly elevated Bilirubin and thymol turbidity and flocculation studies were positive.

Because of the fact that weekly paracenteses were required and the patient was going progressively downhill with hypoproteinemia and nutritional failure, consideration was given to a portal caval shunt. He was treated with a so-called "crash" therapy consisting of intramuscular vitamins, blood transfusions, broadspectrum antibiotics, high supplement diet, and hospitalization. Repeat liver function studies showed the B.S.P. had fallen to 31%, however, the other studies remained abnormal. The total proteins were not improved.

In December, 1958, a side-to-side portal caval shunt was done under hypothermia. At this time free portal pressure before shunting revealed a pressure of 310 mms. of water, pressure in the hepatic portal vein with the portal vessel clamped revealed a pressure of 300 mms. of water, and at the completion of the portal caval anastomosis the free portal pressure had dropped to 90 mms. of water.

This patient is one year postoperative, has gained

approximately 40 lbs. of tissue mass, and has no recurrent ascites. He works full time, sleeps and eats well, and is immensely appreciative of his improvement.

Repeat liver function studies recently revealed all flocculations to be within normal limits. Total proteins 6½ gms. without reversal of the A/G ratio, B.S.P. 18% in one hour.

Case 2—J. O., 58 year old white female. This patient had rather severe G.I. disturbances for a number of years with multiple hospital admissions. X-rays demonstrated possible esophageal varices without other G.I. Pathology. Examination revealed an enlarged spleen and liver which was markedly tender. On several occasions the spleen was felt to be extremely enlarged but seemed to become smaller over a course of several days.

Esophagoscopy revealed a small rosette of esophageal varicosities at the cardio-esophageal junction. Punch biopsy of the liver revealed Laennec cirrhosis presumed to be on a post-hepatitis basis since there was no history of alcohol consumption.

Liver function studies revealed a slightly elevated B.S.P. of 21%, flocculation studies were negative, there was no jaundice. Other laboratory studies were within normal limits.

Because of the large liver and spleen and the presence of esophageal varicosities it was felt that this patient had portal vein obstruction either on the basis of a cirrhotic liver or a portal vein thrombosis. On 9/26/59 a side-to-side portal caval shunt and splenectomy were performed.

Pressure studies at the time of operation revealed a free portal vein pressure of 290 mms. of water, hepatic portal pressure with portal vein clamps was 290 mms. of water, a portal pressure at the completion of the anastomosis was 100 mms. of water.

Surgery was done with the benefit of hypothermia.

This patient has done well since the operation although it is too early to evaluate her from the point of view of esophageal varicosity problems. It is presumed that the rather marked reduction in portal pressure following the anastomosis would obviate the problem of further coronary and esophageal vein dilatation.

Case 3—C. H., 58 year old white male with a long history of alcoholism with progressive ascitic formation and liver function studies compatible with cirrhosis.

The patient was not a problem because of the ascites but because of the enlarged spleen with persistent low white count. It was felt that hypersplenism existed with the associated cirrhosis and increase in portal vein pressure. Esophagoscopy and barium swallow did not reveal esophageal varices. Because of the persistent enlargement of the spleen and hypersplenism as well as cirrhosis it was felt that the patient would benefit from a splenectomy and portal caval shunt. Accordingly, on 8/25/59 a splenectomy and side-to-side portal caval shunt were performed, under hypothermia.

Pressure studies at the time of operation revealed a pressure of 290 in the free portal vein, a pressure of 290 in the hepatic vein with the portal vessel occluded, and a post-anastomosis pressure of 120 mms. of water.

Postoperatively the patient did poorly during the recuperative phase with a poor wound healing, a prolonged ileus requiring intravenous therapy, multiple dressing changes, etc. The patient eventually healed without difficulty however, and has now returned to his work.

DISCUSSION

A brief background and etiology of portal hypertension and cirrhosis is given with emphasis on the role of obstruction as the cause of portal pressure elevation. It is felt that with cirrhosis there develops an hepatic block to the portal circulation and that arterio-venous fistulae do not significantly contribute to the increase in portal pressure. This has been demonstrated by clamping the portal vein in patients with portal hypertension and then measuring the pressures on the hepatic and portal sides of the clamp. Increase in pressure on the hepatic side must indicate obstructive elements within the liver rather than arteriovenous fistulae.

The cirrhotic patient develops a gradual venous obstruction in his hepatic bed with increase in pressures in the portal, splenic, mesenteric, and coronary vessels. When venous decompression occurs through the collateral vessels, esophageal varices result. This shunting of the portal blood decreases the nutrients which reach the liver by the portal circulation, increases the venous pressure in the upper intestinal tract and causes poor motility and probably inadequate absorption from this area. As portal hypertension continues a gradual transudation of fluid high in protein material occurs from the mesenteric venous system and ascites develops. The end result of this is the patient with poor nutrition, low proteins, esophageal varices, and eventually death due to either malnutrition, esophageal bleeding, or hepatic failure.

In the past, surgery of portal caval shunts was limited to those patients who had several episodes of esophageal bleeding and the presence of ascites was often cited as a contraindication. It is our feeling that the patient with portal hypertension due to cirrhosis should be considered as a candidate for surgery and a shunting procedure once the diagnosis has been established. The presence of cirrhosis with or without esophageal varices, but with ascites indicates portal hypertension. A diagnosis of cirrhosis can be made by liver function studies as well as a punch biopsy of the liver. We consider it fortunate that some patients do not develop esophageal varices early in their disease but may have ascites instead. A side-to-side portal caval shunt is the procedure of choice in this condition which by decompressing the portal circulation and hepatic venous bed, significantly reduces the ascitic fluid formation and prevents esophageal varices from occurring. Three cases are presented supporting this point of view.

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Thinning Hair in Young Women

Young women whose hair suddenly begins to fall out need have no fear they will be left looking like actor Yul Brynner.

Drs. William B. Guy and Walter F. Edmundson of Pittsburgh, writing in the February Archives of Dermatology published by the American Medical Association, said the condition is most likely temporary and can be treated successfully.

They said diffuse cyclic hair loss in women is "rather common" and is entirely different from permanent hair thinning that occasionally occurs in women in middle or late life.

"It occurs in transitory episodes, lasting for several weeks usually. The typical patient is a vigorous otherwise healthy woman.

"Needless to say, the emotional overtones in this situation are great. Some men take the state of their hair seriously. Practically all women do.

"It is the source of much perplexity because there is no apparent cause.

"It is probably a physiologic phenomenon. In these people a significant per cent of hairs enter the resting phase simultaneously. When they fall out in a short period of time, the situation causes alarm."

The authors said the hair that falls out usually is replaced by new hair growth. They said they had been able to arrest the process by administering corticosteroid hormones.

Immuno-Pancreatitis

A Preliminary Report

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Acute and recurring attacks of pancreatitis may be caused by food allergy and attacks may be prevented by eliminating the offending food from the diet. It is well to consider this possibility when dealing with this distressing condition.

THE QUESTION of antigenic pancreatitis, as well as that of the broader question of gastrointestinal allergy, is often disputed and frequently difficult of proof;[1,2,3,4,5,6,7,16,18,21,23,25,26,28] yet that it does occur is without question. There have been relatively few reports dealing with allergy of the abdominal organs, probably largely because of their inaccessibility.[16] Interest is therefore naturally aroused as to the seat of the allergic processes and the nature of the reactions.

The exact measurement of allergic phenomena in general remains unknown, although certain facts about it are known. Antibodies, including anaphylactic antibodies, heterophile antibodies, atopic reagins and blocking antibodies are present in the blood of allergic individuals; however, available data do not reveal a constant association of a given antibody with the various allergic conditions. It would seem, then, that the mechanism of allergy cannot be precisely correlated with the antibodies which have been detected to date.[28] Thus, it would appear that allergy can be adduced as the cause of pancreatitis when an altered reactivity of pancreatic tissue can be demonstrated by the induction of one or more allergens.

Read before the Richmond Academy of Medicine.

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CASE REPORT

University of Pennsylvania Graduate Hospital Case Number 236222: 19 year old white female high school student was admitted to the hospital on 12-28-54 because of recurrent attacks of epigastric pain, and was discharged 1-24-55.

Since May 1953, she had been admitted to a neighboring hospital on four separate occasions because of recurrent epigastric pains. The attacks occurred episodically, varying in duration from one week to one month. The present episode began fifteen minutes postprandially 12-17-54, and was associated with a throbbing vertex headache which later localized bitemporally. There was no nausea or vomiting on this occasion; however, all other episodes had had concomitant nausea and vomiting. Vomitus contained bile colored fluid and sometimes recently ingested food particles. The pains were described as cramping or burning in nature, and usually began in the epigastrium with radiation to the left costal margin and left costovertebral angle. Gaseousness and sour eructations were experienced with the pain. There was no relief from changing the position of the body, or from ingestion of Gelusil. Most attacks occurred within thirty minutes after eating provocative foods.

She took three enteric coated pancreatin tablets t.i.d., from August to November 1954 and was asymptomatic for this period of time. She gained thirteen pounds, but had lost most of that during the two weeks prior to this hospitalization. Terramycin was taken in varying amounts with six of her attacks, with apparent relief, and four weeks prior to the present admission she took twenty-four tablets of Terramycin, and again repeated this medication in the week immediately preceding admission.

Until the age of sixteen, she passed four or five large stools daily but since had had only one evacuation daily.

Studies elsewhere had revealed negative cystoscopy, normal retrograde urogram, normally functioning gallbladder without evidence of calculi, normal barium enema, negative flat abdominal film during an attack of pain, several negative studies of the stomach and duodenum.

A progress meal study, however, showed segmentation and flocculation of barium suggesting disordered motor physiology. On two occasions the serum amylase was reported to be 225 mgm.%, but on both occasions blood was drawn on a day when Demerol had been given (50 mgm.). A stool specimen showed slight increase in total fat content.

Past Medical History

The patient was adopted at the age of ten days. At one month of age, the patient had difficulty with her formula and vomited frequently until four months old when Knox gelatin was substituted and seemed to give improvement. She apparently was always rather "sickly" and gained weight poorly as a child. At four years of age, she received a battery of skin tests and "was found to be allergic to many substances". Up to ten years of age, she had been subject to frequent "colds", with tightness in the throat and nasal stuffiness, and had several episodes of "pneumonitis" with an apparent predilection for the right side of her chest. Whenever dogs would come near her, she experienced periorbital edema, rhinorrhea, and would develop a rash. Appendectomy was performed at twelve years of age because of symptomatology very similar to that of the present illness. The appendix was not acute but she had no recurrence of symptoms for five years. There was no history of childhood eczema, hay fever or asthma. Diarrhea was promptly experienced as a baby upon ingestion of pureed peas and lentils. Itching and vomiting were experienced after ingesting veal and pork.

Physical Examination

The abdomen was flat with a right lower quadrant McBurney's scar which was freely movable and nontender. Tenderness was elicited in the epigastrium toward the left end in the left costovertebral angle. Bowel sounds were hyperactive but of normal quality. No organs or masses were palpable. The remainder of the examination was not remarkable.

Laboratory Studies

Red Cell Count	4,350,000	
Hemoglobin	12.5 grams	
White Cell Count	12,400-7,700	
Neutrophiles	64%	46%
Lymphocytes	30%	38%
Monocytes	2%	8%
Eosinophiles	4%	8%
Prothrombin Time	15 seconds/100%	
Sedimentation Rate	8 m.m./hr.	
F.B.S.	85 mgm.%	

Oral Glucose Tolerance Test: Starch Tolerance Tests:

Fasting	96 mgm.%	80 mgm.%
1/2 hour	158 mgm.%	97 mgm.%
1 hour	98 mgm.%	89 mgm.%
2 hours	68 mgm.%	75 mgm.%
3 hours	63 mgm.%	81 mgm.%

Serum Amylase 75 mgm.%—400—290—88—99—320—107

Serum Lipase—0.22—0.61—0.99—1.28—1.0 ccs.

Serum Proteins	6.3 grams %
Albumin	3.7 grams %
Globulin	2.6 grams %
B.U.N.	10 mgm.%
Calcium	9.8 mgm.%
Serum Bilirubin	0.28/0.72 mgm.%

Urinalyses Specific Gravity 1.007, 25 neutral or acid reactions, albumin and sugar negative, sediment negative.

Stool Occult Blood 3+, 4+

Stool Balance Study:

Fat	5.1 gms./3 days
Nitrogen	2.1 gms./3 days
Total wet weight	255 gms.
Total dry weight	44 gms.
Fat content	15.1% of dry weight

Secretin test (80 units I.V.)—Duodenal drainage

Time	*Volume*	*Icterus Index*	*CO2*
0-10 mins.	37 ccs.	250	90 mEq/L
10-20 mins.	10 ccs.	225	95 mEq/L
20-40 mins.	30 ccs.	200	90 mEq/L
40-60 mins.	23 ccs.	250	65 mEq/L
Total	100 ccs.		

Secretin test—Serum Enzymes

Fasting: Amylase 114 mgm.% Lipase 0.53 ccs.

1 hour Amylase 105 mgm.% Lipase 1.14 ccs.

Gastric Analysis: (Ewald test meal) revealed no

free acid in one hour although the total acid reached 27 units at 45 minutes. Bile was present in all specimens. Histalog (50 mgm.) was then given I.M. and within 10 minutes the patient felt a flush and onset of a headache. 20 clinical units of total acid were detected after the administration of Histalog.

X-rays of chest and abdomen were essentially negative. Barium progress meal study showed no abnormality of the esophagus, stomach or duodenum. There was a slight fragmentation throughout the small intestine. The terminal ileum was negative. These changes were ascribed to a disordered motor function but were also considered consistent with the malabsorption syndrome.

Hospital Course

About mid-morning 1-4-55, the patient experienced burning epigastric pains, radiating to the left costal margin and left costovertebral angle. Examination revealed deep left epigastric tenderness, with an area of tympany in the left lower chest and left upper quadrant. No succussion splash was demonstrable and bowel sounds were not remarkable. Blood studies at that time showed a rise in eosinophile count from 4% to 8%. The serum amylase had risen to 400 mgm.% but the lipase remained within normal limits (0.61 ccs.). Urinalysis at the time of this attack was essentially negative. The following day, the fasting serum amylase level was 290 mgm.%. No opiate was administered in relation to this attack. She was treated for pancreatitis by a regimen permitting nothing by mouth, parenteral fluids and electrolytes, Pro-Banthine 15 mgm. every four hours, Gelusil two teaspoonfuls hourly, and hypodermic sodium phenobarbital. This attack continued in varying intensity for three days with gradual subsidence. On the afternoon of 1-6-55, the patient was given four minims of Adrenalin. She experienced tachycardia and palpitations, but soon thereafter was relieved of her symptoms. Serum amylase had already returned to normal limits the afternoon before. On the same evening after the Adrenalin, however, she noted swelling of her hands, and, two hours later, itching of the legs. At this time the possibility of food hypersensitiveness playing a role in the production of her pancreatitis was first considered. Consequently, skin tests for foods, inhalants, and pollen were performed. These revealed strongly positive tests for lima beans, navy beans and suspicious reactions for cauliflower, oysters, egg white, almond, sweet potato, apricot, cashew nut, cotton and house dust. On 1-18-55, a progress meal study was carried out with the barium mixed with chocolate, veal, and lima bean puree as a provocative test. Gastric stasis was noted for three hours, compared with the stomach being empty in one hour previously. The small bowel pattern was essentially unchanged but the patient experienced pain in the umbilical area, with tenderness one half hour after ingestion of the meal. This pain continued until the following day and was associated with itching of the face. The amylase and lipase values were as follows:

Date	Meal	Amylase (mgm.%) Fasting	Amylase (mgm.%) 4 hours	Lipase (ccs.) Fasting	Lipase (ccs.) 4 hours
1-18-55	Barium with veal, chocolate and lima beans	99	320	1.0	—
1-21-55	Veal	91	49	1.1	0.77
1-22-55	Chocolate	55	71	0.48	1.1
1-24-55	Lima beans	72	85	0.62	0.79

In the period between the attacks, which began on 1-4-55 and with subsequent provocative meals, there were no spontaneous episodes of pancreatitis. During this period, she was given a gradual advance in an elimination type diet. Fat and fried foods were omitted from the menu.

The discharge diet was an elimination type and permitted lamb as the only meat initially. Strict avoidance was to be made of all foods in the lentil group, eggs, chocolate, seafoods, gravies, fried foods, orange and pastry. In addition she was given six tablets of triple strength pancreatin with each meal. In the event of, or return of, abdominal pain, nausea, diarrhea, itching, skin rash, nasal stuffiness, etc., she was to record where she had been the previous eight hours and to list all foods ingested the two previous meals.

DISCUSSION

Ivy and Gibbs[2] feel that acute pancreatitis is a relatively common disease which may manifest mild edema without symptoms, or range to a generalized fulminating necrosis with marked pain, shock and, eventually, death. A comprehensive analysis of the possible etiological factors of acute pancreatitis by these same authors lists allergy as a cause of pancreatitis.[10,11,12,13,15]

Some felt that a pancreatic external secretion deficiency, or a surpassing of the pancreatic threshold by excessive total food intake, tends to cause food allergy, and that ingestion of whole active pancreatic extract should overcome this tendency. Markel[15] attributes this deficiency of pancreatic external secretion to "a complicating pathological disturbance of acini following mumps".[30] Boldyreff is generally

accredited as the first to observe, on dogs, that pancreatic enzymes are absorbed from the duodenum into the blood stream.[16,17,18] Walzer reported that the presence of small quantities of protein, sufficiently whole to be identified, are regularly found during digestion, and that these are absorbed into the blood stream. Further, he concluded that in allergic individuals it is the appearance of these whole proteins which produce the allergic phenomenon.

The major allergic phenomena are probably due to release of histamine, or histamine-like substances, into the circulation consequent upon the union of antigens and antibodies. Following this premise, Rodrigues-Olleros[23] was able to experimentally produce pancreatitis in a series of dogs by intravenously injecting histamine. Edema, hyperemia, and hypersecretion of the pancreas were noted. Courelaine and Borgetan[25] sensitized dogs to horse serum by intravenous injection, and then reinjected the serum into the duodenopancreatic vein and directly into the pancreas. Pancreatic edema became apparent at once. Serosanguineous fluid was found in the peritoneal cavity when the animals were sacrificed the next day, and there was evidence of omental fatty necrosis. In addition, the pancreas was edematous and contained many hemorrhagic areas. Maragliano[16] and Bezza[21] have also reported rather similar findings in experimental allergic pancreatitis.

Two recently observed cases of pancreatitis[41] were presumably of allergic origin; however, the question of morphine administration in relation to abdominal pains and the serum amylase levels is not clear. Both patients are said to have developed pancreatitis after ingesting apple and chocolate, both of which had produced abdominal distress previously. Shaffer[1] described a case of allergic pancreatitis, associated with generalized urticaria, which was "completely relieved" by administration of antihistamine.

DIAGNOSIS AND TREATMENT

Since it is rather common knowledge that food hypersensitiveness may occur in the absence of reagins and, therefore, may yield negative skin reactions to the offending allergen, the clinician must rely on other means than skin testing for detecting the etiological agent. An adequate history of possible offending foodstuffs is imperative, even though many suspected foods may have been eliminated from the diet anyway. Whether this is true or not, a food diary is invaluable. This is followed with an elimination diet, beginning with a simple basic diet and gradually adding foods to it. The offending food or foods should be detectable and given to the patient to reproduce the symptoms and signs—and then removed from the diet with a relief of symptoms and signs. This should be repeated several times so as to definitely identify the offending agent.[19,28] Barium studies, with and without the offending foodstuff blended in, may be employed to confirm the diagnosis; serial serum amylases and lipases should also be drawn concurrently with the barium studies, both in the fasting state and at intervals during and after the barium study. Elevated pancreatic enzymes, abdominal pains, and the roentgen pattern of hypermotility of the barium meal through the small intestine (reaching the cecum in one hour or less), segmentation or clumping of barium, slow gastric empyting and hypermotility where the lumen has been diminished may be found singly or all together in such a study. Treatment is avoidance of the offending allergens. Adrenalin, antihistamines, vagus inhibiting drugs, phenobarbital, nitrates and aminophylline may all be used to advantage. Because of the tendency to produce spasm of the sphincter of Oddis, morphine given alone is considered inadvisable. Morphine may sometimes be necessary to control pain, however, for, as yet, there is no satisfactory substitute.[36] Blocking the paravertebral sympathetic nerve fibers carrying pain afferents from the pancreas may be helpful in relieving the acute phase of the pain; however, this may be more useful as a therapeutic tool in later stages of chronic relapsing pancreatitis. Relief of any dehydration or electrolyte imbalance, along with other supportive measures, are also indicated.

SUMMARY

Antigenic pancreatitis is seen clinically, and may be experimentally produced in animals. Food hypersensitiveness, especially chocolate, nuts, seafood and milk, seems to be the chief etiological agent in this condition.

A case illustrating antigenic pancreatitis has been presented.

More information as to the basic nature of the processes involved is needed, and still further investigation of this little explored field should offer rich returns.

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The Problem of Hiatus Hernia

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Hiatus hernia is often overlooked. Even after it is found, it is often difficult to make an accurate evaluation of its role in producing a group of symptoms.

HIATUS HERNIA presents a problem in diagnosis and treatment that engages the attention of many physicians. The general practitioner, the internist, the gastroenterologist, the cardiologist, the surgeon, the hematologist, and the endoscopist are all concerned with the problem. About five per cent of those patients having an upper gastrointestinal x-ray examination have a hiatus hernia.[1] There has been no dearth of recent literature on the subject. However, most of the articles have presented only one phase of the problem or described results in one or more forms of treatment.[2] There has not been a recent article approaching the problem from a broad viewpoint with the proper appreciation for the diagnostic and therapeutic frustration it can cause.

Accordingly, it seems worthwhile to relate the experience of one who has seen personally the vagaries of the condition in forty private office and hospital patients collected between January, 1950, and the fall of 1959. It is acknowledged that no great statistical significance can be put on such a small series collected from among the general run of internal medical patients. It is also true that available statistics have not been of great help in treating some of the patients reported on below. All but four were diagnosed while under my care and, in my role of "family" internist, I have treated them for hernia or other symptoms an average of three and a half years. In some, an accurate diagnosis was not made as soon as it should have been because it was not considered. Various forms of treatment have been used with varying degrees of success.

From the Roanoke Valley Medical Clinic.

CLASSIFICATION AND PATHOGENESIS

Hiatus hernias are of two types: paraesophageal and sliding. In the first type, a knuckle of the fundus of the stomach herniates above the hiatus beside the esophagus. The esophago-gastric opening is usually below the diaphragm. In the second, the cardia of the stomach lies above the diaphragm.

The symptoms in many instances, it would appear, are due to altered pressure relationships and not to stricture, erosion, or esophagitis. In support of this theory, which has not been suggested in the literature or shown experimentally, would be the acute suddenness with which a feeling of pressure in the chest can occur after eating and be relieved just as abruptly. Surely, esophagitis does not become cured in a matter of seconds.

Age, obesity, increased abdominal pressure, as in prostatism and pregnancy, have all been incriminated as important factors. In sixteen of the cases reported here, there was marked dorsal kyphosis although marked obesity was not present. It is mentioned often in the literature that bending over will often cause hernial distress. Perhaps chronic bending, as in kyphosis, has some causal relationship to the hernia.

As in herniae of other types, there is doubtless a congenital predisposition of the ligamentous structures to give way with the stresses of aging but this concept does not lend itself to proof. Trauma has been blamed for the subsequent development of hiatus hernia.

Finally, no one can say in a given instance why a hiatus hernia develops.

THE DIAGNOSIS

There are twenty-two women and eighteen men in the series. Eleven, or about one-fourth, were under fifty years of age. One was a twenty-one year old man. There was a tendency toward corpulency and none of the patients was asthenic in habitus. Only two, however, were grotesquely obese.

The symptoms are protean, variable and intermingled so that they often pose a diagnostic dilemma. They can be classified only generally. The patient may present himself with symptoms which mimic the digestive, cardiac, respiratory, hematologic or purely

functional disorders. He may come in with a mixture of symptoms which only continued observation, sometimes for many months, can elucidate.

The symptoms of hiatus hernia may be so severe as to seem a threat to life or so trivial that treatment is not necessary. The digestive symptoms are mostly those of obstruction or those frequently described lately as being due to peptic esophagitis. They may complicate other gastrointestinal diseases. Eleven of the forty complained that "food would not go all the way down," or that there was a distressing lump which formed at the level of the xiphoid, immediately after swallowing, which would be relieved either by "moving on down" or leaving the table and regurgitating. Such regurgitation was not attended by marked nausea and often the patients could return and eat a full meal. This obstructive symptom had not caused marked weight loss or impairment of health except in one patient. In this group, patients with marked pain are not included. It is believed that this symptom is almost diagnostic, is most easily confirmed radiologically and is of least importance as a health hazard. In a pure form, this lump-at-the-xiphoid syndrome was not aggravated by the supine position, a consideration which has perhaps been over-emphasized. Palmer found symptoms aggravated by recumbency in only 22 per cent whose symptoms were ascribed to hiatus hernia.[3]

Case 1 will illustrate this syndrome in extreme form. A sixty-seven year old, hypertensive, kyphotic woman had had a lump behind her xiphoid, immediately after swallowing, for years. Her eating resembled that of a puking baby—much work for little nourishment. She was embarrassed because she felt she had to eat alone and could not attend church suppers or eat in a restaurant. An operation was mentioned and she readily assented. A large paraesophageal hernia was repaired and she was able to attend church suppers again.

Esophageal pain has been described as burning, boring, frequently radiating into the back or throat, attended by nausea, often worse on reclining and varying from negligible to being so severe as to require opiates.[4] Seven patients in the present series had predominantly symptoms of this type. The main symptoms were mild and yielded to simple treatment but the diagnostic dilemna is illustrated by case 2.

This patient, a seventy-five year old, nervous woman developed epigastric pain in 1950. For years, she had been a finicky eater complaining of gas and heart burn. It was marked by radiation into chest, throat and accompanied by anorexia and weight loss. An upper gastrointestinal series was negative. A cholecystogram revealed a very poorly functioning gall bladder and a cholecystectomy was performed in 1952. Post operatively, she continued to have marked epigastric and burning esophageal pain and anorexia. She was very discontented with her surgery and, in an effort to ameliorate her severe symptoms, she was operated upon for the relief of her "post-cholecystectomy syndrome" in 1956. A sphincterotomy was performed since no other disease was evident. She had been referred by her family physician to a conservative surgeon who, in turn, referred her to me. She was a thin, aged woman, brow-beaten by her husband, and labeled as senile by her children. Thus the stage was set for diagnosing a functional disorder. After months of antispasmodics, sedatives, and antacids, she fell and fractured her hip in 1957. While convalescing, she had an exacerbation of her esophageal symptoms. Another gastrointestinal examination was made and this time a small hiatus hernia was discovered. This patient had had nearly ten years of misery, two major operations and is still not well. The probable cause for her symptoms could have been found earlier had she not had gall bladder disease and, more important, if the esophageal symptoms of hiatus hernia had been fully appreciated.

As shown above, the correct evaluation of atypical symptoms of other diseases may point a shadowy finger to hiatus hernia. This is important because the hernia symptoms frequently occur with other digestive conditions. Two patients had concomitant gall bladder disease. Seven had peptic ulcers of stomach or duodenum. Two had an esophageal diverticulum above the level of the hernia and in one of these a peptic ulcer was found in the herniated portion of the stomach.

The compounding of symptoms and the confounding of the diagnosis can be illustrated by a fifty-eight year old illiterate man who was first seen with a mild case of broncho-pneumonia who was treated as an out-patient. On his return visit, he complained of vague hurting beneath his ribs and in his epigastrium and the inability to eat without vomiting. He was decidedly tender in the right upper abdominal quadrant and cholecystography revealed a non-functioning gall bladder which could account for all his symptoms. However, since he was having marked regurgitation, a gastrointestinal series was done which nicely demonstrated a hiatus hernia. Thus, failure to explain all the symptoms did not result in the treatment failure as described in case 2. A

cholecystectomy and repair of the hernia were accomplished with difficulty in one abdominal operation.

It would appear that the proper appraisal of symptoms referable to esophagus, heart, stomach, duodenum, or gall bladder should, so far as possible, rule out a hiatus hernia. To put it another way, since twelve of the forty of these patients had other digestive or heart disease, it is well not to blame the hernia symptoms on more prevalent and more easily demonstrated conditions.

It is well established that a hiatus hernia can cause symptoms indistinguishable from coronary insufficiency. It is equally well established that hernia and coronary disease can co-exist, making appraisal of either condition difficult or impossible. In this series, there were eleven patients who would have abrupt attacks of retrosternal crushing pain not related to eating. The pain could be so intense as to cause prostration. It would not infrequently radiate into the neck or the left arm. In this series, however, none have resulted in shock or marked tachycardia.

Case 4 illustrates the myocardial infarction syndrome of hiatus hernia. A fifty-eight year old physician was seen at his home about 11:00 P.M. He was moaning with pain, clutching his chest, sighing for breath, and perspiring profusely. Intermittently, he gagged but could not vomit. He did not get relief from 12 mgs. of morphine and was then hospitalized. He was placed in an oxygen tent and finally became comfortable after another dose of morphine. Several electrocardiograms were negative. After twelve hours, he was comfortable. After thirty-six hours, a G.I. series revealed a small hiatus hernia. He has lived eight years since this episode and has no cardiac disease.

Hiatus hernia may mimic milder forms of coronary disease. A fifty-eight year old machinist complained of retrosternal pain when he straightened up from his work and walked fifty yards. The symptoms had been present three weeks and occurred almost daily. The pain was indistinguishable in character from angina pectoris and this diagnosis was made although an electrocardiogram was normal. He was advised to stay at home and rest for two weeks in anticipation of a myocardial infarction. When he returned, he complained of mild indigestion and a G.I. series revealed a very large hiatus hernia and a deformed duodenal cap. An EKG was still normal. On modified ulcer regimen, he has remained well. This case illustrates one strong differential point in distinguishing coronary disease and hiatus hernia: angina pectoris is usually consistent; hiatus hernia is bafflingly inconsistent.

Case 5 illustrates the confusion of co-existent coronary disease and hiatus hernia. This sixty-four year old diabetic woman complained of "upset stomach" in early 1957. She had much belching and sour stomach, with occasional mild nausea. A G.I. series was negative. Two months later, after eating a full meal in a large restaurant where she had walked from a parking lot and climbed a long flight of stairs, she was stricken with severe retrosternal pain radiating into the left arm. She was carried to the hospital via ambulance and treated as a case of myocardial infarction. An electrocardiogram was consistent with the diagnosis. She made a good recovery and has remained relatively well though she still has indigestion. An electrocardiogram remains abnormal but a G.I. series, in May, 1959, revealed a hiatus hernia. She can precipitate chest pain by exertion and still be relieved by nitroglycerin. She is unable to distinguish between the chest pain that comes after eating and that coming after exertion.

Case 6, a seventy-eight year old woman had "heart disease" for twenty years after an attack of infarction syndrome. She had never been truly disabled but she would have attacks of chest pain and dyspnea at night every year or two. Finally, she had a G.I. series which revealed a large hiatus hernia. Digitalis which nauseated her was discontinued, a left phrenic resection was done and she was much improved. Her electrocardigram revealed no marked abnormality. Three "cardiac" cases demonstrate the importance of always considering hiatus hernia in dealing with chest pain. One can only imagine the number of people with hernias who have been misdiagnosed as having coronary disease. One can but speculate on the number of people with cardiac disease whose symptoms are difficult to appraise because a hiatus hernia is unsuspected.

Respiratory symptoms have not presented themselves with any purity in this series. Case 6, above, is described as resembling an attack of paroxysmal nocturnal dyspnea. Several cases of lump at the xiphoid patients stated that it felt as if something "cut off the breath" temporarily but this was not particularly distressing.

Another group of patients pose a special problem that may occasionally be a challenge to the hematologist. Case 7 is such a patient. She was seventy years old and was first seen in 1954 in the hospital where she had been placed for treatment of labyrin-

thitis. Three years previously, she was found to be anemic and was given several blood transfusions. Subsequently, she had been given liver injections, vitamins, and iron but the anemia remained. When first seen, the hemoglobin was 7.5 grams. Gastric analysis revealed no free hydrochloric acid after histamine stimulation. A G.I. series revealed a "very small amount of the gastric mucosa had slipped through the esophageal hiatus producting a hiatus hernia about 2 or 3 centimeters in diameter." Her weakness and anemia again became pronounced and she was hospitalized in May, 1955. A bone marrow examination revealed marked hyperplasia of the erythroid elements. Later, in 1955, she was hospitalized at a University Medical Center where the marrow was reported as very cellular, suggesting chronic blood loss. No other cause for anemia has been found. The fact is that she is still anemic and still going at the age of seventy-six with hematologic findings about as they were six years ago.

Only after several years would one suggest that her anemia, varying between 5 and 10 grams of hemoglobin, was due to bleeding from the hernia. Marked hypochromia and rare early red cells in the peripheral blood and a barely palpable spleen persist. The enlarged spleen is not incompatible with chronic blood loss according to Wintrobe.[5]

The dilemma of diagnosis is presented nowhere more nebulously than in the frank neurotic. Case 8 was a fifty year old, hysterical women first seen in 1951. She had had her left kidney, the gall bladder, and the uterus removed. She had had two laparotomies for adhesions. She had classic hysterical bloating as described by Alvarez. She would have attacks of vomiting and severe epigastric pain, suggestive of gall stone colic. Finally, in 1957, a G.I. series revealed a small inconstant hiatus hernia. She is considered beyond surgical redemption at this time. One can but wonder if she may have had a happier life had her hernia been demonstrated thirty years ago.

I agree with Palmer[3] who poignantly sized up the matter when he stated, "What is needed is a broad clinical formula to permit identification, not of patients with hiatus hernia—for that would be missing the point—but rather of patients with hiatus hernia whose troubles are due to hiatus hernia." In his study of 214 patients, "more than one-third had apparently never had symptoms attributable to hiatus hernia".

Case 9 presents an interesting point. This patient was sixty-one, markedly kyphotic with osteoporosis of the spine. She complained of vague chest pain and indigestion with some regurgitation. She presented herself to a thoracic surgeon. A G.I. series revealed a hiatus hernia and she was operated upon, without relief. She became hostile to her surgeon and hostile to me when I could not offer her immediate relief. When last heard from, she was receiving chiropractic consolation which will give more comfort than her thorocotomy.

These cases are outstanding examples of the problems presented by hiatus herniae. At best, the physician can think of the condition, look for it, appraise it, fit it in with the symptoms, call in all the help he can find from radiologist to endoscopist, and pray he will be right.

TABLE I

Prominent Symptoms in Forty Cases of Hiatus Hernia

"Xiphoid lump"	11 patients
"Esophagitis"	7 patients
"Coronary"	11 patients
"Ulcer"	11 patients
"Anemia"	4 patients
"Functional Indigestion"	14 patients

TABLE II

Concomitant Disease in Forty Cases of Hiatus Hernia

Duodenal ulcer	6 patients
Gastric ulcer	2 patients*
Gall bladder disease	2 patients
Esophageal Diverticulum	2 patients
Symptomatic heart disease	3 patients

*1 patient had gastric and duodenal ulceration.

It is not the purpose of this report to assume any radiological burden but, since the objective diagnosis most often is by x-ray, certain consideration must be taken into account. Not all hernias are demonstrable even by contortions particularly favored by one or another radiologist; not all the hernias found are symptomatic. Some favor a right anterior oblique position, some a left oblique Trendelenburg, some a right oblique prone position, with or without abdominal pressure. One author believes that supine straight leg raising has particular merit. The point seems to be that they are best demonstrated in the position in which the examiner takes the best look. The right oblique prone position, with use of a radiolucent cushion as advocated by Wolf and Guglielmo[6] seems to have particular merit. In this series, the radiologic diagnosis was made by myself in twenty-seven of the cases. As Palmer[7] has pointed out, "the radiologist is not able to detect the junction between the esophageal and gastric mucosa and must render his opinion

on the basis of the number and configuration of the regional mucosal folds, plus the point of sudden increase in the tube's diameter". In any event, the literature indicates they are easily missed. Case 10 is that of a fifty-four year old man who had "sour belches" and intermittent, severe, left chest pain for fifteen years. He had been x-rayed five times, by myself and others, including once at a University Medical Center. Finally, I was able to see a small paraesophageal hernia which was subsequently repaired.

The finding of a hiatus hernia does not mean hernial disease but, where symptoms are highly suggestive, it is worthwhile not to abandon the diagnosis because of a negative x-ray. An occasional hernia will be found by a routine film of the filled esophagus which can be done without special manipulation by having the technician give a swallow of barium about eight seconds before she takes routine stomach x-rays. My experience has been that when a hernia was not demonstrable by the left posterior oblique or the right anterior oblique view, it was not demonstrable by the Trendelenburg position. Certainly, the management of a cup of barium in the dark by enfeebled patients is not easy even without lowering the head.

TREATMENT

The treatment of hiatus hernia poses certain problems. Surgeons are more concerned with eradication of the disease; the internist is concerned with a patient living comfortably with it, if possible. In general, the symptoms are not so severe as to be worth the price in inconvenience, risk and morbidity of a wide thoracotomy. The mortality of operation is about three per cent[8] and I have seen no statistics on the painful post-surgical chest which is a problem to the internist and general practitioner, if not to the surgeon.

Accordingly, a patient with a symptomatic hernia can be told that if his symptoms become severe enough, surgery probably can be of great help but that the surgery has certain hazards. Since the condition is seldom dangerous, the decision can be left to the patient. On this basis, three patients chose thoracotomy. Two of the three got a good result and were satisfied with the operation. One left town soon after the operation and no follow-up was available on her. It is interesting that all three of these cases has paraesophageal hernias, and chose operation. One lady got to the surgeon first and got an unhappy result from her thoracotomy.

Three other elderly ladies were treated by left phrenic resection which had had favorable reports as a paliative procedure.[9] Two had marked regurgitation and one had anemia of abrupt onset for which no other cause could be found. Such a mild procedure has as its purpose a relief of pressure on the stomach fundus and in enfeebled people certainly seems worthwhile. In these old ladies, a good result was promptly attained. They were all markedly kyphotic but in only one was mild dyspnea produced by the diaphragmatic paralysis.

The experience with surgery is related only to emphasize the fact that it is not often necessary. Some of the chest surgeons, nowadays, are becoming so concerned with the esophagitis, which has recently become fashionable, that they are overlooking the three per cent mortality of thoracotomy. Esophagitis is usually quite manageable medically and has an almost negligible mortality.

Seven had some surgical procedure—all of them still need antacids, antispasmodics, or a bland diet from time to time. The remaining thirty-three need antacids, antispasmodics, or a bland diet from time to time. This is not to knock surgery; it is to emphasize that the surgery is no panacea and should be reserved for those whose symptoms are severe enough for them to really be eager for it.

SUMMARY AND CONCLUSIONS

Hiatus hernia is a problem in diagnosis. It frequently mimics or co-exists with other illnesses. At times, its significance is difficult or impossible to appraise. Its presence can confuse; a delay in its diagnosis may lead up therapeutic blind alleys. It produces no symptoms or may produce severe symptoms and its treatment is symptomatic. These are the conclusions one arrives at after considering forty closely observed cases in private practice in the last nine and one-half years.

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Exhibit at University

The University of Virginia Medical School Library is exhibiting memorabilia of Dr. William M. Kerr (or Carr) who entered medical school at the University of Virginia in 1826, one of the first years of the Medical School. Dr. Robley Dunglison was his teacher in anatomy and medicine when only one year was required for graduation and no clinical study was available. Dr. Kerr had studied previously under Dr. John Floyd and at Hampden-Sydney. Dr. Floyd in addition to practicing medicine was a member of the General Assembly, served in Congress from Abingdon District, and from 1830-1834 was Governor of Virginia.

The exhibit shows two of the textbooks which Dr. Kerr used at the University of Virginia. One in Latin, "Medical Dissertations" by Archibald Pitcairne was printed in Rotterdam in 1701. The copy Dr. Kerr owned is initialed by the author and is inscribed to Pitcairne's father-in-law, Sir Archibald Stevenson of Edinburgh. Dr. Pitcairne was so famous in his day and had such a great library that, on his death, the library was bought by Peter the Great of Russia.

The other textbook shown is "The Family Female Physician, or a Treatise on the Management of Female Complaints, and of Children in Early Infancy" by Alexander Hamilton, M.D., Professor of Midwifery in the University of Edinburgh. This book is an American edition, printed in 1793 in Worcester, Massachusetts.

Dr. Kerr emigrated to Indiana in 1831 at 21 years of age to begin his practice of medicine in Dublin, Wayne County. He moved to New Castle after gaining a wide reputation as a physician during a cholera epidemic. His final home in Indiana, after his marriage, was painted in a butter bowl by some itinerant artist. This painted bowl is in the exhibit.

Also shown in a medical lamp intended for the burning of herbs to clear the atmosphere of "vapours". The small bronze statue on this lamp is Mercury with his emblem of the two serpents, the caduceus symbol which is used widely today by physicians. This rate lamp by the Florentine sculptor Giovanni Da Bologna (1524-1608) may also be seen in the Bargello Gallery in Florence, in a larger form.

Dr. Kerr was the great grandfather of Horace Burr of Carrsgrove who loaned this material for the exhibit. The exhibit will be up until early fall.

Aspirin Poisoning – $C_9H_8O_4$

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This is one of periodic articles on common poisonings which will be published in the Monthly from time to time. It is prepared by Dr. Kaye in collaboration with the Richmond Poison Information Center.

ASPIRIN is the most common poison in children between the ages of one to four. It is estimated that more than 100,000 cases are treated in the Emergency Room annually. Aspirin poisoning is especially deceptive in that the child does not always appear too sick in the early stages. Therefore, early recognition and treatment many times may make the difference of survival.

Synonyms: Acetyl salicylic acid, M. P. 137° C., MLD approximately 20 Gms./150 lb. man.

Derivatives: Sodium salicylate, MLD approximately 20 Gms.; Phenyl salicylate (salol), approximately MLD 5 Gms., M. P. 42° C.; Methyl salicylate (oil of wintergreen), approximately MLD 30 ml.; Salicylic acid, approximately MLD 8 Gms., M. P. 157° C.

Uses: Analgesic, antipyretic, antirheumatic.

Remarks: Salicylates are rapidly absorbed and usually can be detected in the urine within 30 minutes. Allergy is not uncommon, usual reaction produces uticaria, angioneurotic edema. The most serious outcome of overdosage is a profound acidosis.

Symptoms: Nausea, vomiting may be delayed; disturbance of hearing and vision, tinnitus. Large doses first stimulate and then depress the central nervous system; initial excitement and convulsions followed by stupor and depression. Central stimulation also produces hyperventilation and an early alkalemia, followed by a severe metabolic acidosis, cyanosis, sweating, thirst, bleeding gums, lowered prothrombin, peripheral vaso-dilatation, hyperpnea with dyspnea, renal and brain damage, weakness and fatigue, hypotension, irritability, delirium and confusion, respiratory failure, collapse, coma, death.

Identification:

(1) To 5 ml. of urine, add 1 ml. of 10% ferric chloride. (First gently heat the urine to rule out possible false reaction due to acetone bodies.) If salicylate is present, a purple color will appear. This test is very sensitive and is positive in urine even after the ingestion of one 0.3 gm. aspirin tablet. This test is also positive for sodium, phenyl or methyl salicylates, and phenol derivatives.

(2) Acidify 5 ml. of blood by adding 0.5 ml. of concentrated hydrochloric acid. Then extract with 10 ml. of ethylene dichloride. Allow layers to settle and separate. Discard upper blood layer, and add 2 ml. of 0.2% ferric nitrate to the ethylene dichloride. A purple color results if salicylate is present. The color can be compared with standards similarly prepared or may be read in a spectrophotometer.

(3) Benedicts' reagent —> positive test for sugar in urine.

(4) Pure crystals can be differentiated with ferric chloride (10%) (1 ml.).

Antipyrine + ferric chloride —> red color.

Salicylate + ferric chloride —> deep violet color.

Amidopyrine (pyramidon) + ferric chloride —> fading light violet color.

Methyl salicylate + ferric chloride —> violet color. (Add alcohol to dissolve methyl salicylate.)

Phenyl salicylate (salol) + ferric chloride —> violet color. (Add alcohol to dissolve phenyl salicylate.)

Phenothiazine + ferric chloride —> lilac color.

(5) Spectrophotometric, Ultraviolet spectra; maximum density; 300 mu. Minimum density; 265 mu.

NOTE: Can be detected in the urine within one hour after ingestion and usually can still be found up to 24 hours or more depending upon quantities taken.

Treatment: Gastric lavage with warm water. Do *not omit* lavage or emesis unless dose is known to be small, or exposure was a *very* long time ago. Patient may appear deceptively well when first seen. Signs and symptoms may be delayed several hours. Vomiting is stimulated centrally and may be delayed.

Fluids, oral or parenteral, to increase urinary output, and to counteract dehydration due to vomiting and sweating. Maintain body heat, fluids, and electrolyte balance. Keep warm and quiet. Alkaline drinks.

Sodium bicarbonate or sodium lactate IV, M/6 to combat acidosis and sodium loss. Periodic blood determinations for alkaline reserve will estimate severity of poisoning and progress of treatment.

Vitamin K derivatives and ascorbic acid for low prothrombin and capillary fragility.

In severe poisoning, an exchange transfusion for children; or hemodialysis (artificial kidney) may prove effective.

Sedation for restlessness. Morphine derivatives are contraindicated! Oxygen and artificial respiration if needed.

Office of Chief Medical Examiner
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Drug Aids Victims of Parkinson's Disease

Tolbutamide, a drug customarily prescribed only for diabetics, has been used with surprisingly good results in the treatment of Parkinson's disease by two physicians.

Writing in the March 26th Journal of the American Medical Association, Drs. Edwin W. Gates, Niagara Falls, N.Y., and Irving Hyman, Buffalo, N.Y., told how the drug had helped 15 persons with Parkinson's disease.

The disease attacks the nervous system causing a stooped posture, stiffness and slowness of movement, rigidity of facial expression, and a tremor of the limbs.

"In some patients there was a decrease in tremor, and in others the most marked effect was the lessening of the rigidity. Most of the patients given tolbutamide were benefited to the extent that they required less care and could perform many more daily living tasks."

The two physicians began using the drug to treat patients with Parkinson's disease after one of them noted a marked decrease in the tremor of the hands and fingers of a 57-year-old diabetic given tolbutamide in 1958.

There is no known cure for Parkinson's disease. Antispasmodic drugs are the usual treatment to alleviate the symptoms. Tolbutamide is a synthetic hypoglycemic agent which lowers the blood sugar level.

The physicians said the significance of their findings is not clearly understood and further research is needed.

Tobacco and Renal Function and Diseases of the Urinary Tract

A Review of the Effects of Smoking and Nicotine Administration in Man and Animals [1,2,3]

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A comprehensive review of the effects of tobacco on renal function in man and animals.

A SURVEY of the world-literature on tobacco has failed to discover any comprehensive review of the effects of smoking on renal function or of the relationship of tobacco to diseases of the urinary tract. This same survey has also pointed up the relative paucity of observations of the effects of nicotine on renal and urinary-tract function in experimental animals; so that a review of the subject at this time may well serve the double purpose of indicating what is known, and what it would be desirable to know. We shall preface our account of clinical investigations and epidemiological surveys on man by a brief summary of the reported effects on the urinary system of animals of acute and chronic tobacco and nicotine administration.

[As used in this review, the term "tobacco" includes all of its preparations as well—tobacco-smoke, nicotine, etc.]

EFFECT OF TOBACCO ON THE MECHANISMS OF RENAL SECRETION IN ANIMALS

Opinion is virtually unanimous that nicotine, tobacco-smoke, or tobacco-smoke solutions cause vasoconstriction in isolated or *in-situ* kidneys; this has been reported for the isolated perfused kidney (Mosso, 1874; Romm & Kuschnir, 1928; Yokoyama, 1938, 1938-39; among others); in the kidney of the dog isolated except for its nervous connections (Sacchetto & Wittine, 1927; Sacchetto & Martinelli, 1927); in the kidney of dogs cross-anastomosed to the circulation of donor animals (Tournade & Malmejac, 1930, 1933); and in the organ *in situ* with circulation and nervous connections intact (Wertheimer & Colas, 1891; Moore & Row, 1898; Marshall & Kolls, 1917, 1919; Gotsev, 1936, 1937, 1940, 1943; Bariéty & Kohler, 1939; Fleig & de Visme, 1907a, b; Tournade & Malmejac, 1930; Roffo & Ramirez, 1932).

It is well known that one of the effects of nicotine administration in animals is a considerable increase in systemic blood pressure. According to Claude Bernard (1857), Werber had reported a diuretic effect of nicotine as a result of its pressor action, and Claude Bernard himself agreed that this would be a reasonable consequence of increase in blood pressure. E. Fischer (1944) studied the effect of nicotine on urine secretion rate in anesthetized dogs, and calculated that the dose which would produce 50% of the maximal possible pressor and diuretic effects was about the same; and J. H. Burn, Truelove & I. Burn (1945) found that hydrated rats given subcutaneous injections of nicotine showed a larger diuresis than the controls during the first 15 minutes after injection, and these workers considered the initial increase in urine excertion to be due to a rise in blood pressure caused by nicotine. E. K. Marshall & Kolls (1917, 1919) concluded from experiments on dogs that intravenous injection of nicotine paralyzed the ganglia of the fibers of the splanchnic nerve supply-

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[3] The material presented has been largely excerpted from a manuscript of a monograph currently in preparation in which the authors deal comprehensively with the world literature on all aspects, experimental and clinical, of the biologic effects of tobacco.

ing the renal vessels. This observation offers a possible, or at least a partial, explanation of the diuretic effect of nicotine. [However, painting the coeliac ganglion with nicotine in dogs and rabbits markedly reduced urine volume (Ellinger & Hirt, 1925).] That the diuretic effect cannot be attributed entirely to changes in blood pressure is evident from the experiments of Fischer (1944), who showed that the rate of urine formation (when this was moderate to begin with) in anesthetized dogs was increased by nicotine after the blood pressure had returned to normal levels from its initial rise; and after large doses of nicotine, this increased secretion lasted for more than half an hour.

The pressor effect of nicotine is due to general vasoconstriction, in which, as indicated above, the kidney participates; and this renal vasoconstriction results in an initial transient anuria (Marshall & Kolls, 1917, 1919). Creatinine-clearance studies performed by Maren (1951) showed a transient fall immediately after injection of nicotine, thought to correspond to a vascular effect during this period.

Fischer (1944) found that the renal effect of nicotine in anesthetized dogs was dependent on the functional state of the kidneys: for poorly-secreting kidneys, the effect was practically nil; if the kidneys were forming urine at a moderate rate, this rate was increased by nicotine; and when the kidneys were already performing maximal work (as in diuresis), the nicotine effect was more or less suppressed. The renal effect of nicotine was not distinctly influenced by anesthetics (Dial or ether). In rats, however, phenobarbital in high (130 mg./kg.) doses inhibited the nicotine-induced antidiuretic effect, but augmented the chloruretic effect, while urethane potentiated the antidiuretic effect, but did not influence the chloruresis (Supek & Eisen, 1953). Phenobarbital in lower (50 mg./kg.) doses had no statistically valid effect on the urine secretion of hydrated rats (Rosenblum, 1954). These results emphasize the importance of performing experiments on renal function on unanesthetized animals.

Pickford (1939) observed that when a water diuresis was produced in an atropinized dog, it was promptly inhibited by the intravenous injection of acetylcholine, and this inhibition no longer occurred after the posterior lobe of the pituitary had been removed. Since the action of acetylcholine occurred in the presence of atropine, it seemed likely that the effect was due to the nicotinic properties of the former drug; hence, J. H. Burn, Truelove & I. Burn (1945) studied the effect of nicotine and smoking on water diuresis; they found that adequate doses of nicotine inhibited diuresis in previously hydrated rats, and this observation has been amply confirmed (Maren, 1951; Pardoe & Weatherall, 1952; Supek & Eisen, 1953; Rosenblum, 1954; Fingl *et al.*, 1955; Bisset & Walker, 1957). Inhibition of water diuresis by nicotine was produced in renal-denervated rats as well as in intact animals (Fingl *et al.*, 1955), but not in hypophysectomized rats given daily injections of anterior-lobe extracts (Burn, Truelove, & Burn, 1945) or in neurohypophysectomized animals (Maren & Bodian, 1951). These latter observations indicate that the antidiuretic action of nicotine depends on the presence of the posterior lobe of the pituitary gland (Burn, Truelove, & Burn, 1945). Although, following administration of nicotine to hydrated rats, the time to reach a maximum rate of excretion was prolonged, the total volume of urine passed was increased in comparison with control animals (J. M. Walker, 1953). The antidiuretic action of nicotine in hydrated rats was not inhibited by ethanol (Fingl *et al.*, 1955; Bisset & Walker, 1957); chlorpromazine (Dasgupta & Hausler, 1955; de Wied & Jinks, 1958); hexamethonium (Walker, 1953; Bisset & Walker, 1957); pentamethonium, tetraethylammonium, diparcol, procaine, or atropine (Supex & Eisen, 1953); parpanit (Supek & Eisen, 1953; Rosenblum, 1954); Artane, diphenylhydantoin, mesantoin, paramethadione, or trimethadione (Rosenblum, 1954). Of the several anti-epileptic drugs tested by Rosenblum (1954), only phenacemide significantly antagonized nicotine antidiuresis. Nicotine also produced an antidiuresis in (presumably unanesthetized) dogs (Maren 1951; Dearborn, Lasagna & Marshall, 1952).

EFFECT OF NICOTINE ON THE COMPOSITION OF URINE

Marshall & Kolls (1917, 1919) stated that one of the most characteristic changes in the composition of the urine after section of the splanchnic nerve in dogs was an increase in chlorides excreted by the kidney on the operated side; but, after intravenous injection of nicotine, this discrepancy in the amounts of chloride eliminated by the two kidneys disappeared. In anesthetized dogs, nicotine caused increased urinary secretion without distinct changes in electrolyte concentration, but during water or caffeine diuresis, nicotine led to an increased electrolyte content of the urine (Fischer, 1944). The antidiuresis in hydrated rats given nicotine was accompanied by chloruresis (Supek & Eisen, 1953;

Bisset & Walker, 1957). The chloruresis might have been due to the release of oxytocin, since several workers have attributed the chloruretic effect of pituitary (posterior-lobe) extract to its oxytocic fraction (see Bisset & Walker, 1957).

Painting the coeliac ganglion with nicotine in dogs and rabbits markedly reduced urine volume and increased total nitrogen and uric acid (Ellinger & Hirt, 1925).

HISTOPATHOLOGY OF THE KIDNEY IN ACUTE AND CHRONIC POISONING

Esser & Kühn (1933) acutely poisoned three guineapigs with nicotine, and found a varying but distinct turbid swelling of the kidney epithelium, its degree dependent upon the time between the injection of the nicotine and death of the animal. The pathogenesis of these relatively uncomplicated acute effects would appear to merit further investigation. Nicotine is readily found in the urine of experimental animals following injection of the drug (Langley & Dickinson, 1890, among many others), and the proportion of administered nicotine excreted unchanged in the urine increases with increasing nicotine dosage (Finnegan, Larson & Haag, 1947); but, so far as the mechanism of urinary excretion of nicotine is concerned, the only published data appear to be those of Ozawa (1929, 1930), who reported that nicotine was eliminated from both glomeruli and tubules in the toad. Investigation of the site of nicotine excretion in the mammalian kidney, and factors affecting this, is greatly to be desired.

Histopathologic changes following chronic administration of tobacco or nicotine are both difficult to report and difficult to assess; the first difficulty is a matter of terminology, the second, of dosage. It appears to us sufficient to record which workers reported finding "pathological" changes in the kidneys of their experimental animals, and which workers found the kidneys to be essentially "normal". In the former category, kidney damage, specified or unspecified, was reported in some or all animals examined following chronic nicotine feeding in the dog (Favarger, 1906), chronic tobacco-infusion injections in the guineapig and rabbit (Benigni, 1911; Perazzi, 1912), and chronic tobacco-smoke exposure in mice (Essenberg, 1952; Venulet & Moskwa, 1952). According to other workers, the kidneys showed no pathological changes following chronic tobacco-infusion injection (Gouget, 1906: rabbit), chronic tobacco-smoke exposure (Schmiedl, 1913: rabbit; Küchle *et al.*, 1952a, b: rat), and chronic nicotine feeding (Wilson, McNaught & De Eds, 1938: rat) or nicotine injection (Nakasawa, 1931, 1933; Werle & Uschold, 1948: rat). It seems probable that too much importance should not be placed on either the "positive" or "negative" results, since it is apparent from a consideration of the entire Tobacco Literature that, if the dosage of tobacco or its derivatives in chronic experiments be too great, non-specific (e. g. nutritional) alterations will be brought about leading to a cachexial state and to general non-specific damage; while, on the other hand, if doses too small be employed, no tissue damage at all is demonstrable, which of course cannot be taken as evidence that no biochemical damage has resulted. Thus, the problem of dosage in chronic animal experiments such as the above, requires a great deal more thought and attention than the earlier workers, for example, have accorded it.

EFFECT OF TOBACCO ON WATER BALANCE

Burridge (1911) found that frog gastrocnemius muscles placed for 24 hours in nicotine-containing Ringer's solution gained 34-38% in weight; the weight gain was independent of nicotine concentration. Frog gastrocnemius dipped in hypotonic salt solution containing 5% nicotine swelled more than muscles dipped into the same fluid without nicotine (Lapicque, 1921). On the other hand, gastrocnemii contracted by 1:2000 nicotine imbibed a smaller amount of water, when immersed in distilled water, than did normal muscle (Gentile, 1934, 1935). The experimental conditions under which these three sets of experiments were performed, however, were so unlike that comparison of the results can scarcely be made. It is of interest to note that the increase in imbibition found by Lapicque was correlated with the decrease in chronaxie observed during the first phases of nicotine poisoning, and that curare, which increased the chronaxie of muscle, decreased its imbibition.

Frogs placed in tobacco-smoke solutions developed a toxic edema, their weight increasing 3-4-fold in 12-24 hours, with ultimate death (Moskwa, 1951). Injection of smoke solution into the lymph sac resulted in 1-2 minutes in marked edema of the viscera. As a result of daily intravenous injection of nicotine, two of five rabbits developed thick, edematous heads comparable to those produced in similar experiments by epinephrine (Leva, 1907). Tainter (1926) reported that paraphenylenediamine injected subcutaneously into rabbits or cats produced edema in

0.75-1.5 hours, and that this could be prevented in the majority of cases by subcutaneous or intravenous injection of nicotine. Other experiments led this worker to believe that the anti-edema effect of nicotine was primarily due to epinephrine secretion.

Injection of nicotine into anesthetized dogs invariably caused an intense secretion of lymph for 5-31 minutes, the increased rate of secretion reaching 500-1100% over control levels; the increased secretion was attributed to intensified peristalsis, increased blood pressure, and accelerated respiratory rate resulting from nicotine (Yaroslav *et al.*, 1940).

EFFECT OF NICOTINE ON THE URETER AND URINARY BLADDER

In a few experiments performed on chloralosed dogs, Bariéty & Kohler (1944) noted that nicotine given intravenously had inconclusive effects on the action of the ureter. Injection of nicotine into the region of the renal plexus of rabbits resulted in acceleration and/or intensification of ureteral peristalsis on the injected side in about 80% of the cases (Cohn, 1928).

Nicotine in a concentration of about 1:1000 stimulated the contractions and increased the tonus of segments and rings of excised pig ureters, while concentrations of about 1:500 paralyzed the contractions (Macht, 1916, 1917; Satani, 1919). Much more inconstant results were reported by Gruber (1927, 1928); in some instances, stimulation was observed, in others, depression of contractions, and in still others, no effect; stimulation was more often observed when the nicotine was added to the solution bathing the pace-maker. Occasionally, nicotine caused reversal of movement, while in a few cases, the drug induced rhythmical contractions in inactive muscles. The effect of nicotine on the motility of isolated pig ureters gradually decreased in going from the lower to the upper end of the ureter (Hasama, 1932); however, action-currents recorded from all portions of the ureters were comparable (Hasama, 1931, 1932). Normally, the currents were diphasic with a frequency of 2-8 per minute, and amplitude and rhythm were uniform in a given preparation. Following application of nicotine and independent of dosage, the action-current was initially increased, but after a time, the amplitude became reduced and simultaneously the frequency gradually decreased, until finally the current fluctuations completely stopped. Nicotine had either no effect or at best only a questionable stimulating one on longitudinal strips or circular sections of upper, middle, or lower thirds of excised dog ureter (Roth, 1917). On ganglion-free isolated human ureters, nicotine stimulated contractions, but further addition of the drug suppressed them (Hryntschak, 1925). The majority of the above workers (Macht, 1917; Satani, 1919; Hasama, 1932) assumed that the action of nicotine was on ganglionic structures in the ureter; but Hryntschak (1925) maintained that nicotine was a direct stimulant to the smooth muscle.

In rabbits, the urinary bladder was said to be unaffected by even large intravenous doses of nicotine (Nasse, 1865), but in the dog, intravenous injection of 10 mg. of nicotine caused marked contraction of the bladder (Henderson & Roepke, 1935), and in the cat, doses as low as 0.5 mg. caused a strong contraction (Truhart, 1869; Langley & Anderson, 1895; Dixon, 1903-04; Elliott, 1906-07; Langley, 1910, 1911-12; Edmunds & Roth, 1920); in the latter species, the contraction was followed by a very considerable relaxation. The effect of nicotine on the nervous control of the bladder has been studied by several workers (Elliott, 1906-07; Langley, 1911-12, 1918-19; Edmunds & Roth, 1920; Lee, 1935; Evans, 1936; Tournade, Chevillot & Bernot, 1938); and the results of such studies, in conjunction with observations in the intact animal of the nicotine effect following administration of atropine (Truhart, 1869; Langley, 1911-12; Edmunds & Roth, 1920), apocodeine (Dixon, 1903-04), and curare (Langley, 1910, 1911-12), and the effects of nicotine on the isolated bladder (Adler, 1918; Sakata, 1928; Lee, 1935; Imai, 1936, 1937; Shimada, 1938, 1939-40), have indicated that the mechanism of action of nicotine in producing contraction of the bladder is primarily by way of the nervous apparatus (Langley, 1910, 1911-12; Edmunds & Roth, 1920; Henderson & Roepke, 1935; Imai, 1936, 1937), although a direct effect of nicotine on the bladder musculature is also a possibility, especially with relatively large doses (Edmunds & Roth; Henderson & Roepke; Imai).

Stimulation of the sympathetic supply to the bladder of the cat at any point in its course produced a rise in the internal pressure of the bladder, followed usually by a fall of much greater extent than the original rise (Stewart, 1899). In some instances, the injection of nicotine seemed to affect the proportion between the rise and the fall. In the rabbit, intravenous injection of nicotine produced a temporary rise in vesicular pressure, followed by a fall (Uchigaki, 1927, 1929).

Truehart (1869) found that urination, which was

a consistent result of large doses of nicotine in cats, was absent in atropinized animals. Langley & Dickinson (1890) noted that brief micturition often occurred on injecting nicotine into rabbits, cats, and dogs.

No gross or histological changes in the urinary bladder were found in rats chronically exposed to tobacco-smoke (Küchle *et al.*, 1952).

We may note here that nicotine is rapidly absorbed from the urethra of rats, guineapigs, rabbits, and dogs (Macht, 1937-38, 1938). Upon injection of buffered solutions of nicotine into the bladder of cats, the absorption and toxic effects progressively increased from pH 4.5-6.0 to pH 9 (Travell, 1940). Travell, Bodansky & Gold (1940) suggested that, when the urine was alkalized, the cat might be poisoned by the nicotine which had collected in its own bladder, and which in consequence of the alkalinization was now reabsorbed, and that a similar poisoning might occur in tobacco-smokers from reabsorption of nicotine from the human bladder when the urine was alkaline. This latter suggestion has been rebutted by Haag & Larson (1942).

EFFECTS OF SMOKING AND NICOTINE ON RENAL FUNCTION IN MAN

It may be recalled that tobacco (taken internally) was once regarded as a very serviceable diuretic (Fowler, 1785; Sigmond, 1838), although long since fallen into disuse. Two early investigators subjected themselves to experiments in which they compared their urine output during control and smoking periods. Hammond (1856) reported that, compared to control non-smoking periods, the quantity of urine and its urea and chloride content were decreased during five-day periods in which six cigars were smoked per day, while the amount of free acid, uric acid, phosphoric acid, and sulphuric acid eliminated through the kidneys increased. Hare (1885) concluded from rather similar experiments that cigarette-smoking increased the quantity of urine. Many years later, Wenusch & Scholler (1935) tested the effect of smoking three or four cigarettes on the urine output of three subjects (two female occasional smokers and one male heavy smoker) under controlled conditions, and from their observations concluded that tobacco-smoke possessed a diuretic effect proportional to the degree of blood-pressure rise resulting from smoking. The diuretic effect was said to be stronger, the less the smoker was accustomed to smoking, although, if acute mild nicotine poisoning occurred, as in one of their cases, the diuresis temporarily ceased.

When subjects are rendered diuretic following the ingestion of water, alcohol, or water combined with an intravenous injection of glucose, however, smoking or nicotine injection is followed by antidiuresis rather than diuresis. This effect—one of the most interesting and sensitive of the pharmacological responses to smoking—was first demonstrated by J. H. Burn, Truelove & I. Burn (1945), and has been confirmed by a number of other investigators; and the conclusion is fully warranted that nicotine inhibits water diuresis through the release of antidiuretic hormone from the posterior lobe of the pituitary gland (review, J. H. Burn, 1951). It should be emphasized that this nicotine antidiuresis occurs only in the hydrated subject or animal, i. e. in the presence of a renal output of diuretic proportions. This is not surprising, since vasopressin [Pitressin] itself is effective only in the presence of an adequate urine flow. This explains the well-known fact that ordinary smoking by subjects in a state of normal water balance (i. e. non-hydrated) has no effect on renal secretion, or, at any rate, no antidiuretic effect. That smoking may on occasion cause an initial and temporary diuresis is indicated by the experiments of Wenusch & Scholler, cited above.

J. H. Burn and his associates (1945) at first considered it surprising that smoking should cause an antidiuretic action in man, if this were due to nicotine, since the amount of nicotine in the smoke was relatively much smaller than the doses producing this effect in animals. Later work, however, enables one to state that the antidiuresis resulting from cigarette-smoking or from injection of similar amounts of nicotine is comparable in degree; for example, G. P. Burn & Grewal (1951) found that urine flow was inhibited for 60-180 minutes following intravenous injection of 0.8-2.7 mg. nicotine, and for 105-195 minutes following the smoking of 1-2 cigarettes.

Considerable individual variation in sensitivity to the antidiuretic effect of cigarette-smoking exists (J. H. Burn, Truelove & I. Burn, 1945; Bisset & Lee, 1958), and the amount of antidiuretic hormone (ADH) liberated by one to two cigarettes also varies according to the sensitiveness of the subject (G. P. Burn & Grewal, 1951). When cigarette smoking was done so intensely that side-effects (pallor, sweating, nausea, giddiness) were experienced by the subjects, the amount of ADH liberated was very greatly increased (G. P. Burn & Grewal, 1951); and the

antidiuretic effect of intravenously-injected nicotine also seemed to be increased in those subjects who had severe and protracted reactions (Hanenson *et al.*, 1956). The antidiuretic effect of smoking appears to be less when tolerance to smoking has developed (J. H. Burn, Truelove & I. Burn, 1945; J. H. Burn, 1957); and, on the whole, smokers have been found to be less sensitive than non-smokers to the antidiuretic effect of cigarette-smoking (J. M. Walker, 1949; Chalmers & Lewis, 1951). Bisset & Lee (1958) have suggested that, in those individuals who do not respond to the antidiuretic effect of cigarette-smoke, release of ADH from the neurohypophysis is inhibited by a high level of epinephrine in the circulation. It is possible to carry this speculation somewhat further to explain the differences in response between smokers and non-smokers. In the doses obtained from ordinary cigarette-smoking, nicotine (1) stimulates the supraopticohypophyseal mechanism to release ADH, and (2) stimulates the adrenal medulla to release epinephrine. The latter hormone, under certain conditions, inhibits the release of ADH; and whether smoking will or will not result in antidiuresis thus depends upon the quantitative relationship between (1) and (2). If smoking has indeed a lesser antidiuretic effect in (hydrated) smokers than in (hydrated) non-smokers, the explanation may lie in one of two alternate possibilities: (1) The adrenal medulla of smokers is more sensitive (less tolerant) to nicotine than that of non-smokers. Or (2) the supraopticohypophyseal mechanism of smokers is more tolerant (less sensitive) to nicotine than that of non-smokers. Since more or less limited tolerance to the general effects of nicotine does develop in normal (i. e. non-hypersensitive) smokers, it is likely that the supraopticohypophyseal mechanism shares this tolerance. In individuals hypersensitive to nicotine, however, it is not impossible that the adrenal medulla shares in the hypersensitivity to the drug, as Bisset & Lee imply. We may perhaps instance the tolerance to the diuretic effect of caffeine which develops in habitual tea-drinkers as an analogous phenomenon.

In one of Burn's studies, he reported failure to produce antidiuresis in a subject from smoking a pipe; instead, there was an increased rate of excretion. Leishman (1951) repeated this experiment on three young healthy non-smokers who, after drinking a liter of warm water, each smoked a new briar pipe as continuously as possible for 15 minutes and swallowed all saliva produced. As compared to control runs without smoking, diuresis was not inhibited.

Lang & Mokry (1954) studied the effect of inhaling the smoke from one cigarette on 25 clinically healthy males aged 20-65 years; of these, 72% showed suppression of diuresis and 64% showed electrocardiographic changes, and, with but two exceptions, the suppression of diuresis correlated with the development of EKG changes indicative of coronary insufficiency or of a local endocardiac anemia. In patients with congestive heart-failure as well as in normal subjects, antidiuresis was produced by cigarette-smoking (Sinclair-Smith *et al.*, 1950) as well as by intravenous injection of nicotine (Hanenson *et al.*, 1956). An antidiuretic response to nicotine injection was also produced in patients with cirrhosis of the liver and in one patient with Addison's disease (Bernstein *et al.*, 1953).

In agreement with the results described in the preceding section on hypophysectomized rats, Cates & Garrod (1951) reported that in eight patients with diabetes insipidus, there was little or no fall in urinary flow when given amounts of nicotine which caused marked antidiuresis in normal subjects. In some of the patients, however, large (4.5 or 6 mg.) amounts of nicotine did induce antidiuresis, considered to be due to liberation of ADH, suggesting that in clinical diabetes insipidus, lesions of the supraopticohypophyseal system are often incomplete. Also, two patients with functional polydipsia (non-smokers) showed a good antidiuresis after 1 mg. of nicotine. Lewis & Chalmers (1951) tested on 11 subjects with clinical diabetes insipidus the antidiuretic effect of smoking one, two, or three cigarettes, and in seven cases found only a slight change in urine flow, compared to normal subjects. In this connection, a singular case reported by Saxe (1870-71) is not without interest. The patient, a 59-year-old man, developed a saccharine diabetes about a year after stopping the use of tobacco, voiding in 24 hours about 10 quarts of urine. Saxe prescribed a diabetic regimen, and the patient remained in quite good health, but complained of dryness of the mouth and throat. Recalling then that the man had stopped the use of tobacco, Saxe urged that he resume its use to see whether his symptoms were related to abstinence from tobacco; the effect was immediate; that night the patient voided only a normal amount of urine, and the diabetic characteristics of the urine completely disappeared.

According to Carter & Simpkiss (1956), mothers of boys with sex-linked nephrogenic (vasopressin-resistant) diabetes insipidus show defective urinary concentration in response to dehydration, and a di-

minished antidiuresis after vasopressin. Garrod (1956) tested one such mother with intravenous vasopressin and nicotine, and found a failure to respond normally to very large doses of vasopressin (i. e. a quantitatively subnormal response), while intravenous injection of 2 mg. nicotine base produced a qualitatively abnormal response: instead of an abrupt antidiuresis, during two hours there was a gradual fall in urine flow from 7.4 to 0.68 ml./minute. Endogenous creatinine outputs indicated a transient reduction in the glomerular filtration rate. Garrod was unable to explain the odd pattern of the nicotine response.

Burn, Truelove & Burn (1945) considered it probable that nicotine, or the nicotine absorbed from tobacco-smoke, stimulated the supra-optic nucleus and adjacent nuclei which also send nerve fibers to the neurohypophysis; and when impulses arrive in the posterior lobe, the antidiuretic hormone is released. This view has been concurred in or accepted by most of the workers mentioned above. The inhibition of diuresis is thus a tubular effect, and this has been demonstrated by measurements of insulin clearances and urinary chlorides (Chalmers & Lewis, 1951); changes in glomerular filtration rates are apparently not involved (Eggleton, 1949; Sinclair-Smith *et al.*, 1950; Cates & Garrod, 1951; Hanenson *et al.*, 1956). Circulatory factors, such as a fall in blood pressure, have been minimized (Cates & Garrod, 1951), and may in fact be ruled out by the observation that such circulatory changes as follow administration of nicotine in the doses used are transient in nature, whereas the inhibition of diuresis persists for 1-2 hours. According to Burn, Truelove & Burn (1945), an intravenous dose of 1 mg. nicotine did not exert a single momentary stimulus to the posterior lobe, but rather one which persisted for 3-45 minutes, and one which was found to be approximated by two intravenous doses of 50 milliunits of posteriorlobe extract given 45 minutes apart. A comparable antidiuresis to that following ordinary smoking of one or two cigarettes occurs following injection of 50-100 milliunits of vasopressin (Walker, 1949; Chalmers & Lewis, 1951).

EFFECT OF SMOKING AND NICOTINE ON THE COMPOSITION OF HUMAN URINE

Sinclair-Smith and co-workers (1950), testing normal subjects and patients with congestive heart-failure, found that the antidiuresis resulting from smoking two cigarettes in 5-7 minutes was not associated with any change in the rate of sodium or chloride excretion; these results were confirmed by Hanenson and associates (1956) in subjects given intravenous injections of nicotine. Contrary findings were reported by Walker (1949) in subjects smoking cigarettes or receiving nicotine intravenously, in whom inhibition of diuresis was accompanied by a rise in total excretion of chlorides; similar results were obtained by Cates & Garrod (1951) in patients with diabetes insipidus receiving large amounts of nicotine, and by Chalmers & Lewis (1951) in subjects deeply inhaling the smoke of one to three cigarettes. It may be noted that the antidiuretic hormone causes a decrease in sodium and chloride reabsorption by the renal tubules, and that the chloride-concentrating ability of the tubules, which is enhanced by ADH, is considerably more sensitive to this hormone than is the parallel water-resorbing power (Silvette, 1940). Therefore, it might be expected, under the proper experimental conditions, that nicotine administration or smoking would lead to an increase in urinary sodium and chloride concentrations.

According to Pouchkine (1890, 1891), smoking 25 cigarettes per day for three days had no effect on the acidity of the urine of seven non-smokers. As a result of personal observation, Kohos (1894) stated that tobacco had no influence on urinary acidity.

Küchle and co-workers (1952b) studied the urine of eight male and one female subjects during a one-week control non-smoking period, a two-week smoking period, and finally an eight-day subsequent observation period; no effects due to smoking were found on albumin, urobilin, urobilinogen, acetone, or acetoacetic acid, and no effect was noted on microscopic examination of the centrifuged solids. Urinary excretion of uropepsin varied so widely during periods of abstinence from smoking and during smoking periods that no conclusions could be drawn relative to the effect or lack of effect of smoking (Cooper *et al.*, 1957).

A few observations have been recorded concerning the urine in nicotine poisoning. Töppner (1931) cited Bucelli (1896) and Stern (1901) as having first observed sugar in the urine as a result of nicotine poisoning. In reported cases of acute nicotine poisoning, concentrations of glucose of about 2% were found (Faulkner, 1933; Joos & Wolf, 1933); but in another case, glucose was absent (Mattei, 1939). More or less albuminuria has been noted in cases of acute nicotine poisoning (Knack, 1932; Willis, 1937; Mattei, 1939), although in another case, no albumin was found (Mattei, 1939).

NICOTINE TESTS FOR NEUROHYPERPHYSEAL FUNCTION

Dingman, Benirschke & Thorn (1957) suggested that the antidiuretic response to nicotine appears to be a useful means of assessing hypothalamic function in man. These workers administered nicotine to nine patients with polyuria to test the integrity of the hypothalamic nuclei, hypertonic saline to assess the function of the osmoreceptors, and pitressin to evaluate the response of the renal tubules to the antidiuretic hormone; the dosage of nicotine employed was 1 mg. in the case of non-smokers and 3 mg. in the case of smokers, given intravenously over a 3-5-minute period. Three patients showed no or little antidiuresis with nicotine or saline solution, indicating a complete loss of or insufficiency in neurohypophyseal function in these diabetes-insipidus cases. In the next three patients, there was a significant antidiuresis following nicotine, but no response to the hypertonic saline; these had posterior-pituitary damage or vascular disease; and the selective failure of osmoreceptor function was postulated to account for the polyuria, since hypothalamic neurosecretory tissue producing ADH was still preserved. The remaining three patients with psychogenic polydipsia demonstrated abnormal responses to nicotine and normal responses to hypertonic saline. Pommatau & Bail (1958) also described studies on the nicotine tests in polyuric and polydipsic syndromes, and they found two limitations which lessen the diagnostic value of the procedure. In principle, a negative test indicates the existence of a true diabetes insipidus; in rare cases, however, the negative results of the test can depend upon a temporary inhibition of ADH secretion. A negative test with a suspected psychic context must always be repeated, if necessary after re-conditioning. In principle, a definitely positive test proves that the supraopticohypophyseal system is intact; however, large doses of nicotine, or normal doses which in some cases produce a secretion of epinephrine, can give a false positive test. Concerning the prognostic value of the test, Pommatau & Bail stated that the importance of an unequivocally positive response in the course of a potomania and, particularly, of a post-traumatic or post-operative polyuria is clearly recognizable, in that it enables one to predict the transitory character of the syndrome. It should be added that the existence of individual variation is sensitivity to the antidiuretic effect of cigarette-smoking illustrated to Bisset & Lee (1958) the unreliability of nicotine or smoking as a test for neurohypophyseal function.

SMOKING AND DISEASES OF THE URINARY TRACT

J. H. Burn, Truelove & I. Burn (1945), noting the antidiuretic action of nicotine and of smoking, raised the question whether those with impaired kidney function should be allowed to smoke. However, Hammond & Horn (1958), analyzing the mortality rates of 187,783 men, concluded that little or no association was evident between cigarette-smoking and nephritis or nephrosis. The case reported by Trillot & Valatx (1947) falls into a separate category; these writers reported a case of subacute glomerular nephritis attributed to tobacco in a man committed to an asylum following alcoholic excess; the patient had been chewing untreated tobacco leaves for several weeks. On the whole, urinary signs and symptoms have been lacking in reported cases of acute nicotine or tobacco poisoning; a frequent necessity to urinate was noted in his patient by Blanchard (1869), and involuntary urination in a fatal case was reported by McNally (1920). McKim, Smith & Rush (1943) reported eight cases of frequency of urination in excessive cigarette-smokers whose urinary symptoms disappeared within 2-3 days after changing brands. The authors considered the cause to be a specific irritating effect of certain chemicals (volatile oils) on the vesical neck, the port of entry being either the lungs or the gastrointestinal tract. In this connection, it may be observed that Jankau (1893) stated that smoking should be denied persons who have undergone bladder operations for the first week after surgery.

An experimental study in mice by Holsti & Ermala (1955) on the subject of oral exposure to tobacco tar and papilloma of the urinary bladder suggested to these workers a relation between smoking and cancer of the bladder. Subsequently, this association has been studied in clinical material, and has occasioned considerable interest and comment (Bonser, 1957; Kennaway, 1957; Brit. M. J. *1:* 218-219, 1957; Study Group on Smoking and Health, 1957; Lancet, Lond. *1:* 1320, 1958).

Lilienfeld, Levin & Moore (1956) demonstrated a statistical association between smoking and cancer of the urinary bladder in men, but not in women. This association was limited to those who gave a history of having smoked for 30 years or more, and was of a lower degree than that found in the case of lung cancer. Denoix & Schwartz (1956) compared 60 men with cancer of the bladder to equal numbers of paired controls (healthy individuals; non-cancer patients; lung-cancer patients), and they concluded

that their data indicated a significant association between cancer of the bladder and cigarette-use, although the association was less pronounced than that which corresponds to lung cancer. This conclusion was somewhat qualified in a later publication in which it was said that an association was observed between tobacco and cancer of the lung, and, probably, the bladder (Schwartz, Denoix & Anguera, 1957). Clemmesen, Lockwood & Nielsen (1958) found a larger proportion of smokers, and of heavy pure smokers of cigarettes, cigars, and cigarillos in 282 men and 87 women with papilloma of the urinary bladder than in the normal population. The increase in frequency of such papillomas among men in Copenhagen was parallel to the increase in frequency of, and mortality from, bronchogenic carcinoma, although occurring at a lower level. These latter authors suggested that it might reduce the risk of recurrences and of malignancy if patients with bladder papillomata abstained from smoking.

In the prospective study by Hammond & Horn (1958) alluded to above, a very high association was evident between cigarette-smoking and cancer of the bladder.

Referring to the studies of Holsti & Ermala and Lilienfeld, Levin & Moore, Bonser (1957) stated that he knew of only one chemical—2-acetylaminofluorine—which, after oral administration, has induced true papillomas and carcinomas of the bladder in mice; he therefore advised caution in accepting experimental data as supporting evidence for the carcinogenic activity of tobacco tar until convincing experiments have been performed. After a review of the paper by Clemmesen and his associates, in addition to the two just mentioned, *The Lancet* (*1:* 1320, 1958) concluded: "An association between papilloma and cancer of the bladder on the one hand and the manufacture, purification, and usage of certain aromatic amines on the other is well established in every country where such an industry exists . . . in this chemical age it is to be expected that the incidence will rise for reasons unconnected with tobacco smoking." *The British Medical Journal* (*1:* 218-219, 1957) concluded that the published data were suggestive of a cause-and-effect relationship between smoking and cancer of the bladder, but that it would be premature to conclude that a relationship had been established; furthermore, it is necessary to explain why no relationship is observed in women, and why cigarettes should be more harmful than other forms of smoking.

We may remark that, contrary to the situation which exists in the alleged smoking—lung-cancer relationship (in which a purportedly demonstrable carcinogen—tobacco "tar"—is known to reach the site of the lesion), no known carcinogen has yet been found in or isolated from the urine of smokers. Since nicotine (and, in all likelihood, its detoxication products) is non-carcinogenic, the presence of nicotine and/or its metabolites in the urine of smokers cannot well be related to carcinoma of the bladder. Benzpyrene, which is on the other hand carcinogenic in animals, is said to be present in cigarette-smoke in concentrations too small to account for pulmonary carcinogenesis (see Wynder & Wright, 1956; Wynder, Fritz & Furth, 1957); and if this is true where the benzpyrene appear to be maximally concentrated, as on the bronchial epithelium, one cannot help but conclude that its concentration in the urine following its absorption and partial or total detoxication and excretion also should be below any "threshold" for bladder carcinogenesis. It would seem, then, that demonstration of the presence of adequate amounts of a known carcinogen in the urine of smokers is a prerequisite to any allegation that there is a "cause-and-effect" relationship between cigarette-smoking and cancer of the bladder. The experiments of Roffo (1931), who placed in the bladder of rats globules of resin in which tobacco extract had been incorporated, and who claimed to have found at the end of 8 months papillary formations in the bladder of a distinctly neoplastic type, are surely irrelevant; moreover, similar lesions were found using distillation products of tar, tricresol, and aniline oil.

THERAPEUTIC USES OF TOBACCO AS A DIURETIC AND IN DISEASES OF THE URINARY TRACT

Fowler (1785) employed tobacco in 31 dropsical cases, 18 of which were cured and 10 relieved. Towards the middle of the following century, a physician signing himself W. A. G. (1840) reported that application of a moistened tobacco-leaf over the epigastrium in two dropsical patients was immediately followed by a profuse discharge of urine and the disappearance of dropsical symptoms.

Pavessi (1853, 1854) reported a case of paralysis of the bladder in an elderly man, which he treated by twice-daily instillations of solutions of nicotine; the bladder acquired a daily increased power of contraction, so that at the end of 15 days, the catheter was laid aside.

EFFECT OF SMOKING ON WATER EXCHANGE IN MAN

In experiments performed on himself, Hammond (1856) reported that, during five-day periods in which six cigars were smoked per day, the amount of aqueous vapor given off in respiration was decreased in comparison with control non-smoking five-day periods.

Sivadjian (1956), using a hygrophotographic method, found that the smoking of two cigarettes by two non-smokers led to an increase in insensible perspiration in the palm of the hand; the number of active sweat glands and extent of glandular activity increased.

This survey of the experimental and clinical literature on the effects of tobacco on renal function and the urinary tract has revealed a curious and even a unique phenomenon. On the one hand, quite massive doses of tobacco or nicotine, acutely or chronically administered, have surprisingly little direct effect on kidney function or structure in animals; and the extensive literature on acute tobacco or nicotine poisoning in man confirms the absence of "acute" effects of tobacco-use on this system, while the exceedingly small number of case-reports of urinary-tract dysfunction or damage in smokers makes it apparent that "chronic" effects of tobacco-use are equally conspicious by their absence. The recent claims of a statistical association between cigarette-smoking and cancer of the bladder would appear to represent a purely artificial correlation; at least, one must consider this to be a strong probability in view of the complete lack, to date, of any biological basis for such a correlation, as well as of any immediately apparent theoretical basis for any such pharmacological-pathological association.

But on the other hand, the effect of tobacco-smoking in releasing antidiuretic hormone from the posterior lobe of the pituitary gland represents one of the relatively few predictable and reproducible pharmacological actions of *ordinary* smoking on the human organism. Although the effects of such ADH release are apparent only in "hydrated", and not in normal individuals, and even so represent indirect rather than direct effects on renal function; yet the susceptibility of the human supraopticohypophyseal system to such small doses of nicotine as obtained in ordinary smoking is a fact of great and interesting importance. Fulton (1955) has stated that the importance of the hypothalamus as a center for integration of visceral function has come to be appreciated only in recent years; and it is to be hoped that future research in the field of nicotine pharmacology (and smoking) will be directed towards the elucidation of the central rather than the peripheral autonomic effects of this drug. Since the hypothalamic mechanisms are particularly sensitive to nicotine, as shown by the antidiuretic effect (potential or actual) of ordinary smoking, it is possible to speculate that the well-known alterations in appetite and body weight, which follow smoking and stopping smoking, are similarly mediated through the hunger and satiety centers which have been shown to exist in the hypothalamus. Thus, a field of research opens up through the observation that the supraopticohypophyseal mechanism seems peculiarly sensitive to nicotine.

Editor's Note: A full bibliography of this report will be used in the reprints which may be obtained from the authors.

Medical College of Virginia
Richmond, Virginia

Pre-Paid Medical Care....

Edited by
RICHARD J. ACKART, M.D.

The Past Five Years

What has happened to Blue Cross and Blue Shield during the past five years is of interest not only as history, but as a portent concerning the future. Perhaps the future of voluntary prepayment as a whole, as well as that of the service Plans—and thus the continued success or defeat of our voluntary, free enterprise system of providing health services—is presaged by the trends which apparently have developed 1955 through 1959.

During the five year period, the Blue Shield Plan which has its headquarters in Richmond, through changes in rates charged, arranged that each subscriber increase by 76% his payments into the pool of public funds which the Plan administers. Throughout the period the Plan's payments to physicians remained quite consistently at 90% of subscriber income; thus, the 76% greater payment made by the subscriber all went to physicians.

The additional payments to physicians, in behalf of each subscriber, covered certain new services which were added to the Blue Shield program and covered an increase in the subscriber's rate of usage of doctors' services, as well as the expense of an upward revision of the Plan's fee schedules. However, the latter change produced a 33-1/3% increase in the amount of payment to a doctor—per service.

Nonetheless, despite the larger payment per service received by doctors, the ratio of adequacy of Blue Shield payments to doctors' charges showed no change during the five year period. In 1959 the Plan's payments related to doctors' charges as follows:

70.3% of surgeon's charges covered
80.4% of medical charges covered
61.5% of consultation charges covered
73.4% of anesthesiology charges covered

These percentages vary from those pertinent to each of the five years by no more than a few tenths of one percent—despite the 33-1/3% increase in Blue Shield fees paid.

In 1955, the Richmond Blue Cross Plan was charging Group-enrolled families $4.90 per month. During 1959 the family rate was established at $9.40, a 92% higher rate. About 28% of the 92% can be accounted for by increases in hospital operating costs, which went up approximately 5% per year throughout the five year period. Thirty-three cents of the 1959 rate, or 3.5%, was needed to cover additional Blue Cross benefits, primarily extending coverage of nervous and mental care from ten days to sixty days. The balance of the 92% rate-hike was necessitated by the progressive increase in utilization experienced during the period.

How local usage of hospital beds has increased is all too glaringly apparent from the following table in which admissions and total days are presented in terms of "per thousand persons covered":

Local Experience

	1955	*1959*
Admissions:	149	165
Average stay:	7.61 days	8.32 days
Total Days:	1090	1370

Thus, the total days of hospital care paid for by the Plan for each 1,000 of its members increased 25.7% during the five year period. At about $22.00 per day, and with 400,000 members, this increase in utilization rate amounted to an increase in Blue Cross expenditures of two and a half million dollars.

Though some increased utilization was to be expected because of medical advances and certain demographic trends, the 25.7% increase of bed-utilization rate in Virginia seems entirely out of keeping with the comparable 7.2% increase experienced by Blue Cross Plans nationally. The discrepancy between local and national experience seems all the more inexplicable when it is considered that the levels of utilization in Virginia in 1955 were higher than those of the nation in 1959.

National Experience

	1955	*1959*
*Admissions:	131	139
Average stay:	7.45 days	7.60 days
*Total Days:	973	1043

* Per 1,000 members

Although the increase in local utilization rate does

not seem based on medical necessity, or even propriety, it is nonetheless explained in large part on the basis of availability of hospital beds.

During the five year period under consideration, the number of hospital beds available for use by local Blue Cross members and their doctors increased 13.2%. If, in fact, the availability of beds increases injudicious utilization, Blue Cross is in for more trouble because as of today, either under construction or on drawing-boards, there are enough additional beds to bring about a further increase of 16.5% by the end of 1962.

Obviously the trends that are adversely affecting the efficacy of Blue Cross-Blue Shield are equally affecting health insurance and other forms of prepayment. The deductible and co-insurance features of prepayment coverage must, of course, be increased —to the detriment of prepayment's ability to do the job the public demands of it. The public's demands will be served, either through its voluntary institutions or through its government. But perhaps it is not too late for the medical profession to take truly aggressive action to preserve the voluntary, free-enterprise way of meeting the public's demands. However, continuance of the laissez-faire policy which the profession seemingly adheres to with a Pollyanna-optimism is tantamount to an invitation for ambitious social-planners to assume the leadership the profession now is failing to exercise.

What to Do in Case of Poisoning

Handy reference cards telling what to do in case of accidental poisoning and how to prevent such emergencies have been made available by the American Medical Association.

The cards reflect a growing awareness of the magnitude of the problem, according to Bernard E. Conley, Ph.D., secretary of the A.M.A. Committee on Toxicology.

Statistics show that 1,422 persons, 355 of them pre-school children, were killed in one year by packaged chemicals, such as detergents, cleansers, and other household cleaning agents, while thousands were injured.

"The first aid for poisoning chart serves as both a reminder of the dangers of accessible poisons to small children and as a help in meeting poisoning emergencies," Dr. Conley said. "Its life-saving potential may be equated with its value in educating the public on what to do when poisoning occurs."

As another means of preventing poisonings, the A.M.A. is supporting a bill, now before Congress, that would require household, commercial, and industrial products to carry labels listing their contents.

The cards suitable for attaching to medicine cabinets, may be obtained free of charge from the Committee on Toxicology, American Medical Association, 535 N. Dearborn St., Chicago 10, Ill.

Public Health . . . .

MACK I. SHANHOLTZ, M.D.
State Health Commissioner of Virginia

"Control of Communicable Diseases in Man"

The rules and regulations contained in "Control of Communicable Diseases in Man", a publication of the American Public Health Association, were adopted many years ago by the Virginia State Board of Health and were declared by this Board to have the force and effect of law within this Commonwealth, except where any town or city, in accordance with authority granted in its charter, has adopted other rules and regulations not contrary to law. This authority has been vested in the State Board of Health of the Commonwealth of Virginia by Section 1487 of the Code of Virginia and by the provisions of other acts.

A new edition, the ninth, of this manual, Control of Communicable Diseases in Man, published early this year, has been received in the central office of the State Department of Health, and is being distributed to the local health departments and to the reporting physicians of the State. Each local health department office will be sent three copies, one for the director, one for the nurses, and one for the sanitarians, and a copy will be sent to each physician who is listed as sending weekly report cards of communicable diseases to local health departments.

A revision of the manual is made each five years by the Subcommittee on Communicable Disease Control, which is currently a subcommittee of the Committee on Evaluation and Standards of the American Public Health Association. This subcommittee requested and received collaboration from the following national and international organizations:

U. S. Department of Defense
U. S. Office of Civil & Defense Mobilization
American Academy of Pediatrics
Conference of Public Health Veterinarians
Ministry of Health for England and Wales
Department of Health for Scotland
Pan American Sanitary Bureau
World Health Organization

This is the fourth edition in which the Ministry of Health for England and Wales and the Department of Health for Scotland have collaborated, and the third in which the World Health Organization has participated.

The aim of the manual is to provide an informative text for ready reference by public health workers of official and voluntary health agencies, to include physicians, dentists, veterinarians, sanitary engineers, public health nurses, social workers, health educators, and sanitarians; and for physicians, dentists, and veterinarians in private practice having a concern with the control of communicable diseases. The booklet does not substitute for standard text books. While primarily intended for workers in the Americas, and therefore largely expressive of American practices and ideas, the content is broadly representative of global infectious diseases.

The report of the committees was originally designed as an aid in formulating standard regulations for administrative control of communicable diseases for which notification was required by State and municipal health authorities of the United States; succeeding editions have given a more comprehensive coverage of world infections. The present list includes most conditions of public health importance, with a goodly representation of the less common and less significant diseases which so often present puzzling problems in administrative action.

Notification to the local health authority that a case of communicable disease exists within the particular jurisdiction is the first step toward control. A system of reporting functions at four levels. The first is collection of the basic data in the local community where disease occurs. The data are next assembled at district, state, or provincial level. The third level is collection of total information for the nation. Finally, for certain prescribed diseases, report is made by the national health authority to the World Health Organization.

The local health department uses the information immediately to tabulate what diseases are within its bounds and for investigation and control. It forwards these reports to the State Department of Health. This office compiles a report of diseases in the State and returns a weekly morbidity report to the local health departments and to the reporting

physicians. It makes a weekly report to the National Office of Vital Statistics and, in turn, receives from that office a weekly report of communicable diseases in the States and Territories of the nation with comments on certain reports of unusual or special interest. Our national health authority reports certain diseases to the World Health Organization, which returns a report of these diseases as they exist throughout the world.

Diseases to be reported to local health departments are:

- Amebiasis
- Anthrax
- Aseptic Meningitis
- Botulism
- Brucellosis (undulant fever)
- Cholera
- Diphtheria
- Encephalitis
  - infectious
  - arthropod-borne
  - other
- Hepatitis
  - infectious
  - serum
- Leprosy
- Leptospirosis
- Malaria
- Measles
- Meningococcal Infections
- Plague
- Poliomyelitis
  - nonparalytic
  - paralytic
  - unspecified
- Psittacosis
- Rabies in Animals
- Rabies in Man
- Relapsing Fever
  - louse-borne
- Rheumatic Fever
- Rocky Mountain Spotted Fever
- Salmonellosis
  - all types including typhoid and paratyphoid
- Shigellosis
- Smallpox
- Streptococcal Infections including scarlet fever
- Tetanus
- Trichinosis
- Tuberculosis
  - all forms
- Tularemia
- Typhoid Fever
- Typhus Fever
  - endemic
  - epidemic
- Venereal Diseases
  - Gonorrhea
  - Syphilis
    - primary
    - secondary
    - early latent
    - other forms
- Whooping Cough
- Yellow Fever

Case report of a communicable disease provides minimal identifying data of name, address, diagnosis, age, sex, and date of report for each patient and in some instances for suspects. The date of onset and the date of diagnosis are useful.

Reports of Epidemics. In addition to requirement of individual case report any unusual or group expression of illness which may be of public concern is desirably reported to the local health authority by the most expeditious means. This procedure is to be followed whether the disease is well known and subject to routine report, indefinite or unknown, or absent from the list of diseases officially reportable. Pertinent data of the epidemic report include number of cases, within what time, approximate population involved, and apparent mode of spread. The report is forwarded to the next higher jurisdiction by telephone or telegraph. Examples of epidemics involving diseases not usually reportable are food outbreaks and influenza.

Diseases should be reported by the physician in attendance on the case. In the event that no physician is called to see the patient, the lay attendant (father, mother, guardian, husband, wife) is responsible for reporting the case.

In addition to his responsibility for the clinical diagnosis and treatment of cases of communicable diseases, the practicing physician has other responsibilities related to the public health of the population or area he serves:

(1) He should report promptly all cases of diseases which are herein declared reportable

(2) He should cooperate with the local and State health departments in carrying out health regulations, especially in instituting isolation and, rare instances, quarantine, and in obtaining release cultures when applicable

(3) He has an additional obligation to cooperate with the local health authorities in developing and executing a good program for the control of communicable diseases

(4) He should acquaint himself with and make appropriate use of the services of the Laboratory of the State Department of Health to assist him in arriving at an accurate diagnosis of illnesses among his patients

(5) He should be familiar with the drugs and biologic products which are provided by the Department of Health free of charge and those for which a charge is necessarily made.

Diseases in the manual "Control of Communicable Diseases in Man", are listed in alphabetical order and under each disease are numerous headings, such as—

- Identification
- Occurrence
- Infectious agents
- Reservoir and source of infection
- Mode of transmission
- Incubation period
- Period of communicability
- Susceptibility and resistance
- Methods of Control:
  - A. Preventive measures
  - B. Control of patients, contacts, and immediate environment
    1. Isolation
    2. Concurrent disinfection
    3. Terminal disinfection
    4. Quarantine
    5. Immunization of contacts
    6. Investigation of contacts and source of infection

7. Specific treatment

C. Epidemic measures

D. International measures

This manual is not distributed generally by the State Department of Health. It goes, as stated above, only to health departments and reporting physicians. Others who may want a copy can order the same from the Book Service of the American Public Health Association, 1790 Broadway, New York 19, N.Y., sending payment in advance. The paper edition costs $1.00 per copy and the cloth bound edition sells for $2.50 each.

MONTHLY REPORT OF BUREAU OF COMMUNICABLE DISEASE CONTROL

	April 1960	April 1959	Jan.-April 1960	Jan.-April 1959
Brucellosis	6	2	11	7
Diphtheria		1	7	3
Hepatitis (Infectious)	70	29	362	164
Measles	1238	3549	3735	8972
Meningococcal Infections	8	9	26	40
Aseptic Meningitis	1	—	10	—
Poliomyelitis	0	0	0	1
Rabies (In Animals)	34	15	104	65
Rocky Mt. Spotted Fever	0	0	0	2
Streptococcal Infections	910	1180	2993	4651
Tularemia	0	0	20	6
Typhoid	0	2	3	6

Leprosy Control

A member of the World Health Organization's Second Expert Committee on Leprosy said that segregation is still needed to control the disease. In a letter printed in the April 30th Journal of the American Medical Association, Dr. H. W. Wade, Culion Sanitarium, Palawan, Philippines, criticized a news release issued by WHO headquarters in Geneva last August.

He said the release to the effect that the committee had recommended abolition of leprosariums is "simply not true".

Furthermore, he said, the statement in the release saying that the age-old custom of keeping lepers apart from the rest of the community is unnecessary from the medical point of view is "hardly less misleading."

"The recommendation of the committee, in keeping with resolutions of several international meetings held in recent years, is that leprosy should be treated more or less as tuberculosis is treated, with hospitals for the care of patients suffering from the acute 'reactional' episodes of leprosy, for the surgical reconstruction of patients with mutilations, and for the training of personnel and other administrative purposes."

Dr. Wade also clarified the committee recommendations in connection with legislation.

"It was recommended that special laws applying solely to leprosy be abolished and that the disease be taken care of by regulations set up under general laws pertaining to the control of communicable diseases as a whole. That would not, by any means, preclude isolation of some patients if it seems necessary under local conditions in the light of present knowledge of the disease."

Current Currents

HEALTH CARE OF AGED: Both political parties seem determined to bring about the enactment of some form of Forand type legislation having to do with health care of the aged. It is important, therefore, that each member know the thinking of AMA on the question and be able to answer the questions which are sure to be asked by many patients.

In commenting on the Administration's recent proposed medical care plan for the aged, Dr. Louis M. Orr, President of AMA, stated that it is based on the false premise that almost all persons over 65 need health care and cannot afford it. This, according to Dr. Orr, is not true. The truth is that a majority of our older people are capable of continuing a happy, healthy, and in many cases productive life. Only 15 per cent of the 15 million persons in the nation over 65 are on old age assistance. An undetermined number, although able to finance other costs, find it difficult to withstand the additional burden of the cost of illness. None of the Forand type bills are tailored to meet these problems.

Here is AMA's positive 8-point program for the health care of the aged.

(1) The needy aged. These aged now receive health care through OAA programs. Here the need is for better organized medical care programs including improved preventive medical care;

(2) The near-needy. This is the group whose size is indeterminate, who can meet ordinary costs of living but cannot pay for health care costs. The AMA supports a state-administered program of federal grants-in-aid to the states for the liberalization of existing OAA program so that the near-needy could be given health care without having to meet the present rigid requirements for indigency. A liberalized definition as determined locally would permit an expanded program and encompass the near-needy group;

(3) Facilities. Better nursing home facilities for the long-term care of the aged person, especially those over the age of 75, is the most urgent health care need before the nation today. The average age of nursing home patients is 80, and their average duration of stay is 2 years. It is here that major improvement can be brought about. AMA supports federal programs for the provision of grants through the Hill-Burton mechanism to provide for new nursing home additions to existing hospitals. For proprietary nursing homes the AMA supported the recently enacted amendment to the Federal Housing Act providing for government guaranteed mortgage loans to proprietary nursing homes. AMA is also cooperating with the American Nursing Home Association

and the American Hospital Association in an effort to bring about a rapid improvement in medical care provided in nursing homes;

(4) Voluntary health insurance. Health insurance and prepayment policies tailored to meet the needs of the aged for long-term nursing home care, must be developed as rapidly as possible. Health insurers and the Blue Cross and Blue Shield Plans across the nation are already experimenting in this new area of coverage;

(5) Home nursing care. Care of the aged patients at home is psychologically, medically and financially desirable. Many programs to promote home nursing care are being developed.

Homemakers services also provide opportunities for children caring for aged mothers or fathers to continue gainful occupation. They need to be expanded;

(6) Attitude toward aged. A basic change in attitude toward the aged person must be brought about. The person who reaches 65 has not suddenly become non-productive, and senescent. On the contrary, most persons over 65 are reasonably well and able to work. Increased productivity by eliminating compulsory retirement and permitting voluntary change of work is an essential part of the answer to the present problem;

(7) Health education. Many older persons are unaware of the need for continuing healthful nutrition and other practices that contribute to good health. Above all the "will to live" is essential to continuing health. Preventive medical measures instituted long before the age of 65 also can contribute materially to the promotion of good health after age 65;

(8) The purchasing power of the dollar. One of the principal economic problems of the aged person in the last twenty years has been the constant and continuing erosion of the purchasing power of his pension benefits. Any government program to help the aged must be anti-inflationary and maintain the purchasing power of fixed pension and annuity benefits.

PILOT EXAMINATIONS: Effective June 15, 1960, student and private pilots must receive their medical examinations from designated medical examiners. Because of the many questions that have been raised concerning this ruling, Dr. James L. Goddard, Civil Air Surgeon, has emphasized that any physician may make application for designation as an examiner. He has requested that your editors publish the following objectives which the ruling hopes to achieve: (1) To maintain a group of medical examiners who are clearly responsive to the needs of public safety in the performance of examinations and the issuance of medical certificates to airmen; (2) To permit the administration of training programs to maintain the quality of performance of medical examiners and to permit the dissemination of special instructions pertaining to the needs of civil aviation; (3) To bring into the program those physicians who have the professional qualifications and a demonstrated interest in the medical certification field.

Mental Health....

DIETRICH W. HEYDER, M.D.

Psychiatric Consultation to Social Agencies

The Mental Health Center of Norfolk has recently extended to the various agencies of the city and county a program of psychiatric consultation. The idea in itself is not new. However, since our experience has been rewarding, we feel that a presentation of our program is warranted.

The task of the Mental Health Center in Norfolk is manifold. While we give diagnosis and treatment to mentally sick individuals, an attempt is also made through general educational efforts to improve the public's opinion of, and emotional reaction to, mental illness and their psychiatric treatment. We also conduct research into the sociological and psychodynamic causes of the diseases with which we work and make some effort at prevention. To these standard services we have added that of consultation to other social agencies and found it to be both idealistic and practical.

The specialized skill and knowledge of our psychiatric team is utilized by the workers of the other agencies in helping the multitude of people with whom they deal. By offering consultation to the various social agencies in the community we can multiply the effects of the Mental Health Center to a considerable degree.

The clinic staff finds the consultation sessions with the persons of different training and experience stimulating. The clinic is now receiving fewer inappropriate referrals for psychiatric workup, and we have understood a great deal more about where to refer problems that we cannot handle. The spirit of cooperation brought about between the Center and the other agencies by this method makes working with them more pleasant, both from a standpoint of personal relations and professional satisfaction.

There are these three facets in our consultation service. First, the professional skills of the psychiatrist, the psychiatric social worker, and the psychologist are made available to the agencies. Second, a program is set up to broaden and deepen the academic knowledge of the personnel of the agencies served. The third is that our own Mental Health Center staff learns from the other agencies what community resources are available in Norfolk.

The decision to provide this broad type of agency consultation was followed by a thorough discussion of how to do it. In the past, certain information had been given to all the agencies at a collective meeting. But there was a feeling that we should provide individually scheduled time for each agency. On the other hand, some agencies were not convinced that consultation would be of benefit to them at all. Others felt that mass meetings offered little in the way of solid achievement because of the extreme differences in the agencies themselves. Agencies differ in size from one worker to over a hundred, services varied from financial assistance to long-term counseling, extensive variations were noted in the amount and type of training the workers had received, while ideological standards (bases on religion or personal opinions) caused differences in recommendations for similar, even identical problems. On the other hand, it was recognized that agencies could gain something from each other. We reached a compromise whereby every agency would be given individual consultation, and a joint meeting of all agencies was planned to follow each period of rotation.

Under the auspices of the Norfolk Council of Social Agencies all agencies of the Community served by the Center were invited to participate. The executive director of one of the agencies was appointed to schedule and coordinate the service.

Consultations for individual agencies were scheduled for each Wednesday from 10 until 12 o'clock, usually in our office. (We have made an effort, however, to visit the office of each agency at least once.) The first consultation with each agency was used to obtain a detailed explanation and interpretation of its functions, goals, and limitations. The second meeting carried the emphasis of what constitutes a psychiatrically valuable history, as well as general referral procedures, plus a restatement of the aims and limitations of a clinic such as ours.

After these preliminaries, the consultation hours were organized and now operate in a rather set man-

Abstract of paper presented to Annual Meeting of Department of Mental Hygiene and Hospitals in Roanoke, November 17, 1959.

DIETRICH W. HEYDER, M.D., *Director, Norfolk Mental Health Center.*

Approved for publication by Commissioner, Department Mental Hygiene & Hospitals.

ner. First, the social history of a selected case is presented by a member of the social agency. This is criticized and discussed thoroughly as to content or lack of content, leading to interpretation of statements made by the client and of his motivation for treatment with stress on the dynamic forces behind the problem at hand. In the interplay between the members of the presenting agency and our staff, we try to arrive at a clear understanding of the case as a whole and in detail.

Of course sessions with an agency fortunate enough to have highly skilled workers, along with a training program for students, differ greatly from sessions with an agency hiring no worker with training in the sociological or psychological field. The level of discussion is adopted to the degree of knowledge.

A discussion of the patient's history and discussion of the handling of the client by the worker frequently leads to criticism of the worker by his co-workers and consultants. Often the very fact that a given case is chosen for presentation indicates adverse feelings between the worker and client. The exploration of these feelings naturally provokes anxiety and hostility on the part of the person who presents the case. If no way is offered to ventilate these feelings, "negative transference" can develop which may even spread to negative feelings and misunderstandings between the agency and the clinic. Therefore, an opportunity on the part of the agency to criticize the consultants is offered freely and often demanded. In this respect the consultative hour can well be compared with the psychotherapeutic hour, where the patient's (agencies) expression of feelings are encouraged and "ventilation" occurs.

The organization asking for consultation is not "advised". Authoritative pronouncements as to the solution of a problem does not usually help. Only what can be worked out by the involved persons is valid and worthwhile. Even so, recommendations can be given and accepted without resistance or resentment if they are based on awareness of the dynamics operating within the client, the recognition of the realistic situation, and the knowledge of the agency's own limitations—limitations that an agency is oftentimes unaware of or does not want to admit possibly because of painful guilt feelings.

The inter-agency meetings, as distinguished from the individual agency consultations, have been of particular interest. They have fostered and improved the inter-action between the consulting members of the Council, where the give-and-take process was particularly invigorating. This might be illustrated by presentation of a case.

One of the family agencies had become involved with a married couple and their three children, one from the father's and two from the mother's first marriage. Behavior difficulties on the part of the parents and the children arose from the immature attitudes of both parents toward their own children. It took a lengthy and complicated discussion including Freudian postulations to clarify part of the history presented and arrive at a workable and dynamic formulation. An unusual amount of criticism was aimed at the psychoanalytic interpretation, a criticism which sharpened the discussion. Eagerness and enthusiasm and emotions ran high. It struck almost like lightning from the blue when a member of the less psychoanalytic minded services arose and asked the very practical question, "Well, what are we going to do about it?" A burst of laughter followed the question together with a sigh of relief, for we all suddenly realized that we were not psychoanalysts but psychiatric consultants and people looking for consultation. We then went ahead with the recommendations.

Generally the discussions are aimed at training personnel, no effort is spared to explain all questions in detail regardless of repetition.

Although the results of the consultation service described above cannot be expressed in statistics, they should be self-evident. Most of the teaching cases presented at the Center can be disposed of without further workup, which saves valuable diagnostic time. The agencies have grown more confident in their own capabilities. The quality of their referrals has consequently improved. They have on the other hand learned to recognize difficulties beyond their scope. They have been able to improve their case work through increased understanding of their clients. The close-knit personal relationships between the Center and the agencies have made all of them feel freer to call on the members of the staff for advice and guidance at any time. This reflects in better service to certain clients who can now receive competent help within a shorter time.

The Center has thus profited in many ways. Cooperation between the Center and the agencies has grown. Through our greater knowledge of the functions and limitations of the agencies themselves the quality and number of referrals to other agencies has improved and the Mental Health Center has become more efficient in the utilization of community resources.

The Medical Society of Virginia . . . .

Council Minutes

A special meeting of the Council of The Medical Society of Virginia was called to order by Dr. Allen Barker, President, at 1:00 P.M., on Sunday, February 28, 1960, at Society Headquarters. Attending were Dr. Guy W. Horsley, Dr. Mallory S. Andrews, Dr. Harry J. Warthen, Dr. Mack I. Shanholtz, Dr. John T. T. Hundley, Dr. Thomas W. Murrell, Jr., Dr. Fletcher J. Wright, Jr., Dr. William N. Thompson, Dr. Byrd S. Leavell, Dr. W. Fredric Delp, and Dr. Richard E. Palmer. Also in attendance were Dr. Vincent W. Archer and Dr. W. Linwood Ball, Delegates to the AMA, Mr. John Duval and Mr. William Miller, Attorneys for the Society, and Dr. Harry C. Bates, Jr., Chairman, Committee on Aging. Also meeting with the Council were Dr. Edward E. Haddock, Dr. Dean B. Cole, and Dr. Benjamin W. Rawles, Jr., all members of the Society's Legislative Committee.

The President informed Council that an unusually large number of bills with medical implications had been introduced in the General Assembly and the meeting had been called for the express purpose of considering them.

First to be considered were House Bills 156 and 503 which had to do with the construction of homes for the aged on a state-local matching appropriation basis. Mr. Thomas Manning, President of the Virginia Nursing Home Association, was introduced and stated that in his opinion the legislation was socialistic and that every effort should be made to keep the State out of the nursing home field.

After further consideration, the following resolution was adopted:

> Be It Resolved, that The Medical Society of Virginia go on record as being opposed to the entrance of the State into the Nursing Home field and that the Governor's Commission on the Aging be urged to continue its studies of existing conditions in this field in order that private enterprise can be properly informed and advised concerning present and future needs.

Council then heard a report by Mr. Duval concerning bills which were of particular interest to the Society and its committees. These had to do with legislative safeguards for committees, amendments to the maternity hospital law, radiation hazards, and medical examination fees and subjects. It was learned that all five bills were progressing satisfactorily.

Next to be considered were those bills pertaining to milk control and supervision. It was moved that Council reaffirm previous actions by the House of Delegates and Council that members of the Legislative Committee in the General Assembly be urged to support House Bills 622 and 623 (would place responsibility for milk control and inspection under Department of Health). The motion further requested that a VALC study be supported if House Bills 622 and 623 were not passed.

Senate Bill 315 was then discussed at great length. This Bill would amend the Virginia Code in such manner as to permit organizations such as the Group Health Association of Washington to operate in Virginia. When Council learned the Legislative Committee had recommended that such legislation be opposed, it adopted a motion to support the recommendation to the fullest extent possible and that the following resolution be used, should the need arise:

> Resolved By the Council of The Medical Society of Virginia:
>
> (a) That a physician owes his allegiance to his patients, and the intervention of a third party between the physician and his patient is inimical to the best interests of the patients, the physicians, and the public.
>
> (b) That the furnishing of medical services by a corporation is contrary to the long established laws and principles governing the practice of medicine in Virginia and should not be permitted.
>
> (c) That the proper function of a medical care program is to assist in the financing of medical costs and not the furnishing of professional services.
>
> (d) That Senate Bill 315 is violative of these established principles and should not be enacted into law.

Next to be considered was Senate Bill 269, which had to do with the labeling of blood for transfusion purposes. Although it was believed the Bill was not popular and quite probably would be killed, it was decided that Council should endorse the following

resolution recently adopted by the Virginia Society of Pathology.

> WHEREAS, there exists no discernable or scientific evidence that blood from an individual of one of the racial groups of human beings differs in any significant quality from individuals of corresponding blood types in other racial groups, and
>
> WHEREAS, there is no evidence that transfused blood persists permanently in any individual, and
>
> WHEREAS, any requirement that blood banks separate and classify blood according to the racial group of the donor would drastically decrease the possibilities of keeping supplied of the types of blood on hand for all therapeutic needs, and tremendously increase the waste of human blood, and would make impossible the use of blood from such important sources as the American Red Cross, and would frequently result in the impossibility of obtaining sufficient supplies of blood of the proper type for the treatment of ill persons:
>
> BE IT RESOLVED, that the Virginia Society for Pathology recommends that the Legislature of Virginia not give favorable consideration to any measure such as Senate Bill 269 which would impose upon the operation of blood banks any unnecessary classification of bloods by scientifically unjustifiable criteria such as the racial groups of the donor.

There followed considerable discussion concerning House Bill 533, which would remove the Virginia Hospital Service Association (Blue Cross) from under the jurisdiction of the State Corporation Commission as far as rate fixing is concerned. The following resolution was adopted:

> RESOLVED, that the Council of The Medical Society of Virginia endorses House Bill No. 533 and favors its passage by the General Assembly of Virginia.

It was then moved and adopted that House Bill 716 be opposed. This was the so-called Christian Science vaccination bill and would have amended the Code in such manner that vaccination would not be required of any child if an affidavit could be furnished stating that such vaccination conflicts with the religious teachings of the individual.

Mr. Miller then explained a proposed amendment to House Bill 328 which would serve to clear up any misunderstanding concerning the methods of treatment (physical therapy) denied to masseurs and others not qualified as physical therapists. Council felt that the amendment was a good one and that no particular action was necessary.

A question was then raised concerning House Bill 198 which would, under certain conditions, make it possible for paying patients to bring suit against charity hospitals. A motion was adopted which would instruct the Legislative Committee to oppose the Bill should such action become necessary.

Dr. Bates asked if a bill establishing 20 General Practice Clinics under the State had been introduced. Dr. Shanholtz indicated, that to his knowledge, no such bill had been introduced.

Dr. Bates then stated the American Medical Association had requested that his Committee on Aging take the initiative in organizing a joint council on aging in Virginia. It was moved and adopted that Council approve efforts on the part of Dr. Bates and his Committee to bring about the formation of such a joint council.

A report by the Executive Committee concerning the physicians award proposed by the A. H. Robins Company was presented by Dr. Wright. He stated that the Committee was of the opinion this would not be a good move at this time, particularly in view of the fact that the Society had just recently abolished the General Practitioner of the Year Award. There was some concern that the award, although proposed with the best of intentions, might very easily do more harm than good. It was Dr. Wright's recommendation that the proposal of the Robins Company be rejected in a most courteous and appreciative manner and the reasons be explained to Mr. Velz. A motion to this effect was adopted.

Mr. Duval then acquainted Council with a bill, supported by the State Hospital Board, which would make it possible to regulate and supervise homes operated by private parties for mentally deficient children. Dr. Shanholtz described some of the conditions he had personally observed at these homes and supported the contention that some authority over them was needed. It was explained that there was known opposition to the bill.

Dr. Archer then reminded members of the Council of the meeting in Washington on March 24 with Virginia's Congressional delegation.

There being no further business, the meeting was adjourned.

ROBERT I. HOWARD, Secretary

APPROVED:

ALLEN BARKER, M.D., President

Miscellaneous . . . .

Mail-Order Rx Schemes

It may well be a long time before the patient who just walked out of your office with an important prescription in his pocket ever gets around to taking the needed medication—the reason—mail-order prescription schemes.

Now being heavily promoted all across the nation, these schemes center their publicity on cut-rate prescription prices.

Here, for example, is what might happen to your patient.

Instead of taking his prescription to his community pharmacist to be filled that day, he drops it into the mail to one of these operations perhaps a thousand miles away. From seven to ten days later, the filled prescription comes back, perhaps too late to do the job for which it was originally intended.

Your patient in the meantime, still experiencing the symptoms which prompted his visit to you in the first place, attempts to tide himself over until the prescription arrives by applying some sort of self-medication which may give him temporary relief and at the same time lessen his understanding of the importance of your diagnosis and the drugs you prescribed.

Not only are dangerous delays characteristic of the mail-order mechanism, but the public is also denied the complete services it has a right to expect from any pharmacist in any pharmacy. As a regular practice mail-order operators refuse prescriptions containing narcotic drugs. Most patients have no knowledge of what is and what is not a narcotic drug; and therefore any warnings by the mail-order house that it will not accept prescriptions for narcotic drugs become meaningless. The patient, after exposure to unnecessary delay, discovers that his prescription is only obtainable from his community pharmacy.

Prescriptions for narcotics are not the only ones which mail-order houses refuse to dispense. There are classes of prescriptions which are being refused by mail-order operators for their own convenience, especially prescriptions which require compounding. Both the ethics and traditions of the pharmaceutical profession demand that a pharmacist make every effort possible to promptly dispense every prescription he receives regardless of the amount of professional attention required.

Because these depots operate on an impersonal assembly-line basis in a jurisdiction where only the supervision—rather than the actual dispensing—by pharmacists is required, the danger of dispensing errors is increased. Moreover, by operating outside the states in which the prescription was originally written, these houses deny the patient the protection he has a right to expect from his own state laws governing the practice of pharmacy.

Another area in which the public is being misled is illustrated by quoting from a full-page advertisement which a District of Columbia mail-order operator inserted in *The Philadelphia Inquirer* of Sunday, April 3, 1960:

> "YOUR PRESCRIPTIONS ARE COMPOUNDED BY REGISTERED PHARMACISTS!
>
> "Our registered pharmacists compound your prescriptions in ultra-modern, regularly inspected pharmacies—using the finest, freshest nationally known ingredients.
>
> "YOUR ORDER IS FILLED IMMEDIATELY TO ASSURE YOU OF FRESHNESS!
>
> "The same day we receive your prescriptions—our pharmacists fill them and mail them out. This assures you of the freshness of every medicine you receive from us. And, of course, with our volume business and volume turnover, our shelves of vitamins and chemical ingredients are constantly replenished with fresh items."

After the publication of this advertisement, it was revealed that the premises described consisted of nothing more than an empty room without any inventory, fixtures, or pharmaceutical equipment.

This advertisement was refused by two other large circulation newspapers, and the matter has been called to the attention of the Federal Trade Commission.

Perhaps as important as anything else to the medical profession is the fact that such schemes make it convenient for certain kinds of practitioners, unauthorized to prescribe in their own states, to write prescriptions that will be filled by these distant operations. The geographical separation between prescriber and dispenser makes it virtually

impossible, and impractical for these operators, to check the source or to offer professional advice to the patient when he receives his medication.

The greatest opportunity for mail-order promotion has been the geriatric market, where public attention has been mostly concentrated in recent months.

A host of other unanswered questions comes to mind when prescriptions are ordered from these assembly-line outfits. For example, how can we safely assume that long-distance dispensing will be as the prescriber intended? Is the way paved for substitution? Has the prescription been compounded under sanitary conditions?

The District of Columbia, where standards for pharmaceutical practice are based on an antiquated 1906 act of Congress, has become the haven for mail-order operators.

One of these Washington-based firms was recently brought before Corporation Counsel of the District of Columbia. The investigating officer described the premises as "without facilities for compounding prescriptions", and noted further that the only "sink was located in the rear of the store by going through a room that had waste paper for wrapping and packaging all over the place and the room where the sink was located proved to be filthy and the sink itself was corroded and stained and in a filthy condition."

That these mail-order prescription schemes are truly a menace to public health was emphasized by a statement made by Dr. George M. Fister, a member of the American Medical Association Board of Trustees, in an address to the recent meeting of the American Pharmaceutical Association House of Delegates. Physicians, he noted, can easily warn their patients of the many flaws in these dangerous mail-order operations. He went on to condemn the mail-order schemes as "one of the gravest problems" facing the health field today.

A physician for more than 30 years, Dr. Fister said that he does his best to discourage any patients from sending prescriptions to a mail-order operation.

"The personal touch is still essential", he said. America's high standard of medical care was "founded on this firm foundation of personal service, and we would be foolish indeed not to preserve it."

The development of highly concentrated synthetic drugs, purified natural products, potent dosage forms and special needs in storage and dispensing all have created new problems of drug supervision, regulation and administration. The pharmacy and drug laws of each state are designed to protect citizens from the effects of ignorance and incompetency in these matters which are beyond individual patient control.

The American Pharmaceutical Association has launched an educational campaign to inform all members of the medical profession about the inherent dangers in mail-order prescription operations. The national professional society is also supporting H.R. 10597 which will bring to the District of Columbia a modern pharmacy act.

At an emergency meeting in Washington, D. C., haven of the prescription-by-mail operators, Dr. William S. Apple, APhA Secretary, declared that, "If this personal pharmacist-patient relationship is not preserved, the next break in the chain will inevitably be the elimination of the physician as the diagnostician and substitution of the mail-order purveyor as the prescriber. The only call on the physician will be to write the death certificate. Individual acceptance of professional responsibility by all members of the medical team can quickly put an end to the development of centralized mail-order prescription depots."

If mail-order prescription hazards are to be fully recognized, the medical profession must join with other members of the health team by reacting quickly and in a positive manner. Otherwise the public will be misled into believing that an impersonal centralized mail-order method is an acceptable substitute for sound community medical-pharmaceutical service.

Book Announcements....

Books received for review are promptly acknowledged in this column. In most cases, reviews will be published shortly after the acknowledgment of receipt. However, we assume no obligation in return for the courtesy of those sending us same.

Textbook of Otolaryngology. By DAVID D. DeWEESE, M.D., Clinical Professor of Otolaryngology, University of Oregon Medical School, Portland. And William H. Saunders, M.D., Associate Professor of Otolaryngology, The Ohio State University College of Medicine, Columbus. The C. V. Mosby Company, St. Louis. 1960. 464 pages with 354 illustrations. Price $8.75.

A Traveler's Guide to Good Health. By COLTER RULE, M.D., New York City. Doubleday & Company, Inc., Garden City, New York. 1960. 266 pages. Price $3.95.

Massage Manipulation and Traction. Edited by Sidney Licht, M.D., Honorary Member, British Association of Physical Medicine, Danish Society of Physical Medicine, and the French National Society of Physical Medicine. Elizabeth Licht, Publisher, New Haven, Connecticut. 1960. xiii-275 pages. Illustrated. Price $10.00.

Your Heart. A Handbook for Layman. By H. M. MARVIN, M.D., Associate Clinical Professor of Medicine, Yale University School of Medicine, etc. Doubleday & Company, Inc., Garden City, New York. 1960. 335 pages. Price $4.50.

One Out of Four. A Personal Experience with Cancer by MYRTLE WILLIAMSON. Foreword by Lewis C. Sharman, M.D. John Knox Press, Richmond, Virginia. 1960. 77 pages. Price $1.50.

The Reluctant Surgeon. A Biography of John Hunter. By JOHN KOBLER. Doubleday & Company, Inc., Garden City, New York. 1960. 359 pages. Price $4.95.

Cancer of the Cervix. Diagnosis of Early Forms. In Honour of Prof. C. Kaufmann. Ciba Foundation Study Group No. 3. Editors for the Ciba Foundation G. E. W. Wolstenholme, O.B.E. M.A., M.B., M.R.C.P., and Maeve O'Connor, BA. Little, Brown, and Company, Boston. 1960. viii-114 pages. With 27 Illustrations. Price $2.50.

Drugs of Choice 1960-1961. Walter Modell, M.D., Editor, Director, Clinical Pharmacology and Associate Professor of Pharmacology, Cornell University Medical College; Attending Physician, New York Veterans Administration Hospital; etc. The C. V. Mosby Company, St. Louis. 1960. 958 pages. Price $13.50.

The History of Medicine. By HENRY E. SIGERIST. Edited and with an Introduction by Felix Martí-Ibanez, M.D., Professor and Chairman, Department of the History of Medicine, New York Medical College, Flower and Fifth Avenue Hospitals, New York; Editor-in-Chief, M D Medical Magazine. Foreword by John F. Fulton, M.D. M D Publications, Inc., New York. 1960. xviii-313 pages. Price $6.75.

Neurosurgery. Volume II. Surgery in World War II. Medical Department, United States Army. Prepared and published under the direction of Major General S. B. Hays, The Surgeon General, United States Army. Editor in Chief, Colonel John Boyd Coates, Jr., MC. Editors for Neurosurgery, R. Glen Spurling, M.D., and Barnes Woodhall, M.D. Associate Editor, Elizabeth M. McFetridge, M.A. Office of the Surgeon General, Department of the Army, Washington, D.C. 1959. xxvi-705 pages, Illustrated. For sale by the Superintendent of Documents, U. S. Government Printing Office, Washington 25, D. C. Price $7.50.

The Clonal Selection Theory of Acquired Immunity. By SIR MACFARLANE BURNET. The Abraham Flexner Lectures 1958. Vanderbilt University Press, Nashville. 1959. xiii-209 pages. Price $5.00.

A Doctor Enjoys Sherlock Holmes. By EDWARD J. VAN LIERE. Vantage Press, New York. 1959. 141 pages. Price $3.00.

Doctor Strand. By BORIS SOKOLOFF. Vantage Press, New York. 1960. 205 pages. Price $3.50.

The Cigarette Habit. A Scientific Cure. By ARTHUR KING. Doubleday & Company, Inc., Garden City, New York. 1959. 96 pages. Price $2.00.

A Practical Guide for General Surgical Management. By JULIAN S. STERLING, M.D., Sc.D., Assistant Professor of Surgery, Graduate School of Medicine, University of Pennsylvania; etc. Foreword by Herbert R. Hawthorne, M.D. Vantage Press, New York. 1959. 67 pages. Prices $3.00.

The Teen-Age Years. A Medical Guide for Young People and Their Parents. By ARTHUR ROTH, M.D. Doubleday & Company, Inc., Garden City, New York. 1960. 288 pages. Price $3.95.

The Story of Dissection. By JACK KEVORKIAN, M.D. Philosophical Library, New York. 1959. 80 pages. Illustrated. Price $3.75.

Medieval and Renaissance Medicine. By BENJAMIN LEE GORDON, M.D., F.I.C.S. Philosophical Library, New York. 1959. xii-843 pages. Illustrated. Price $10.00.

Hope Deferred. By JEANETTE SELETZ. Vantage Press, New York. 1959. 546 pages. Price $4.50.

Editorial....

University of Virginia Hospital 1901

The University of Virginia Hospital (1901-1951)

MRS. SARAH S. MATTHEWS of Charlottesville has just published an excellent volume describing the first fifty years of the University of Virginia Hospital. The author was admirably fitted to write an account of the hospital and those who worked in it for she served as Record Librarian in this institution from 1937 until her retirement a few years ago. Few things transpire within the confines of a hospital that do not receive a mental note by those who work within the Record Room. Mrs. Matthews' retentive memory, happy literary style and lively sense of humor combine to make this a very readable book.

Fortunately the writer did not confine her account to the first half of the present century for the question immediately arises in the mind of the reader as to how a medical school, established in 1826, when Professor Robley Dunglison arrived from England, could exist for 75 years without an associated hospital. Mrs. Matthews devoted the first four chapters to a description of this largely didactic period when the medical student's chief contact with the sick was obtained in an out-patient department. This theoretical type of institution necessitated a hospital appointment after graduation from the University. Fortunately the instruction received in those early years during the prolonged medical session, which began prior to the middle of September and continued, with the exception of Christmas day, until the first of August, was of such quality as to make these graduates sought after by the larger Northern hospitals.

A Dispensary was built in 1895/96 where the George Rogers Clark monument now faces Main Street just East of the C & O Railway overpass. The Medical Department's limited budget would not permit the erection of this building until the discolored bricks salvaged from the Annex and Rotunda fire, the preceding fall, were made available for this purpose. Occasional surgical operations were performed in this building but there were no beds available for in-patients and it was necessary to transport post-operatives across Main Street to a rented building where they were housed during their convalescence. The four students who carried the stretcher were known as "Dr. Buckmaster's pallbearers". Here it was that Dr. Paul B. Barringer, who succeeded

Dr. James Lawrence Cabell as Professor of Surgery, enucleated Col. John S. Mosby's left eye which had been injured in a runaway horse accident.

All this was changed when Dr. Barringer, then Chairman of the Faculty, interested several citizens in donating funds for the first and central wing of the present hospital. Lady Nancy Astor, then Mrs. Shaw, was the first substantial contributor when she raised $600 by making a levy on all of her husband's business friends who incautiously boasted about their financial coups in her presence. The site chosen for the hospital was the former faculty pasture where professors were permitted to keep the family cow. The new 25 bed hospital with operating amphitheater, nurses quarters, kitchen, dining room, storage space and offices cost $50,000 and was formally opened on April 17, 1901. The present hospital and the entire Medical Department have grown up about this modest building.

Mrs. Matthews described in detail the various efforts to merge the clinical facilities of the University of Virginia and the Medical College of Virginia. Needed progress was delayed at the University pending the outcome of this contest but when the decision was reached in February, 1922, not to move the Medical School, the way was opened for the rapid expansion which is still taking place in the Department of Medicine. Additions have been added to the hospital with increasing frequency and the size and quality of the faculty, as well as the student body, have kept pace with the growth of the physical plant.

The last 10 chapters are devoted to the faculty, with short biographical notes, the auxiliary services, the administration and, most readable of all, a series of humorous sketches dealing with some of the oddities that graced the local scene during the half century she described. This story deserved to be told and it has been well told by Mrs. Matthews. Fortunate is the institution that has a recorder of her caliber.

HARRY J. WARTHEN, M.D.

Society Activities . . . .

Roanoke Academy of Medicine.

The April meeting of the Academy was held in conjunction with the thirty-third Annual Congress of Gill Memorial Hospital. Following the business, a very excellent scientific program was presented. Dr. J. R. Heller of the National Cancer Institute of Bethesda, Maryland, presented an address on Concepts of Cancer Research. The second paper was by Dr. Alton Ochsner of Oschner Clinic and Foundation Hospital of New Orleans, Louisiana, on Increasing Menace of Cancer of the Lung.

Having these two eminent authorities and erudite speakers on the same program afforded the Academy and the members attending the Spring Congress a delightful scientific meeting.

Dr. William H. Kaufman is president of the Academy and Dr. James G. Snead, secretary-treasurer.

Richmond Academy of Medicine.

The J. Shelton Horsley Memorial Lectureship was held on April 26th. This was the 14th annual lectureship which was established by Dr. Guy W. Horsley in honor of his father. The speaker was Dr. Oscar Creech, Jr., William Henderson Professor of Surgery and Chairman of the Department of Surgery of Tulane University School of Medicine. His subject was Chemotherapy Treatment of Malignancy.

The Fourth District Medical Society

Held its Spring Institute at the Southside Community Hospital, Farmville, on April 5th. Dr. Austin I. Dodson, Jr., and Dr. Solomon Papper, Medical College of Virginia, conducted a symposium on renal disease.

Virginia Pediatric Society.

The Annual meeting of this Society was held at The Greenbrier, White Sulphur Springs, West Virginia, in February. The following officers were elected: President, Dr. O. Watts Booth, Newport News; vice-president, Dr. John T. Walke, Roanoke; and secretary-treasurer, Dr. Gayle G. Arnold, Richmond.

News . . . .

New Members.

Since the list published in the May issue of the Monthly, the following members have been admitted into The Medical Society of Virginia:

Werner Anthony Gergen, M.D., Front Royal
Michael Homa, M.D., Arlington
Solomon Papper, M.D., Richmond
Nicholas George Poulos, M.D., Richmond
George Straughan Schools, M.D., Richmond
Thomas H. Webb, M.D., Portsmouth

Dr. Vincent P. Hollander,

University of Virginia, has been made a lifetime professor of cancer research through a grant from the American Cancer Society. He will do full time research on cancer, his special interest being the hormonal control of tumor growth. This is the first full time cancer research professorship ever granted by the Society. Dr. Hollander is associate professor of internal medicine and assistant professor of biochemistry, in addition to being coordinator and director of the cancer research laboratories at the University.

Dr. Charles W. Scott,

Burkeville, is the recipient of the Douglas Southall Freeman Award for outstanding work in tuberculosis control. He is superintendent and medical director of the Piedmont Sanatorium. The award was presented at the annual meeting of the Virginia Tuberculosis Association in April.

Scientific Exhibit for Clinical Meeting.

Application forms for space in the Scientific Exhibit at the Washington, D. C., Clinical Meeting of the American Medical Association, November 28 to December 1, are now available. They may be procured by writing directly to Charles H. Bramlitt, M.D., Director, Department of Scientific Assembly, American Medical Association, 535 North Dearborn Street, Chicago 10, Illinois. Applications close on August 1.

The "Hull" award will be presented for the first time at this meeting to the best exhibit on a scientific subject which has not been previously shown at a medical meeting. The award will consist of a gold medal and an honorarium of $250.00.

Dr. Paul D. Camp,

Richmond, attended the meeting of the Pan American Medical Association in Mexico City. On May 3rd, he presented a paper on Serum Cholesterol Distribution in Subjects of Various Ages and Clinical Conditions.

Officers of Virginia Tuberculosis Association.

Dr. E. C. Drash, Charlottesville, has been named first vice-president of this Association, and Dr. John A. Mapp, Nassawadox, third vice-president.

Dr. Fletcher D. Woodward,

Charlottesville, has been made the first honorary member of the Speech and Hearing Association of Virginia.

Dr. Donald L. Brummer,

Richmond, has been elected president of the Trudeau Society of Virginia. He is medical director of the E. G. Williams Hospital.

The American Cancer Society

Has announced that a grant in the amount of $43,350.00 for research in whole body radiation has been made to the Medical College of Virginia. Part of the grant, $25,000.00, will be used toward the purchase of a two million volt x-ray machine. The balance will be used in research activities carried out with the equipment.

The Medical College is receiving other assistance in the purchase of the equipment, including a substantial sum that has been raised by members of the Virginia Federation of Woman's Clubs. The cost of the equipment is approximately $95,000.00. Construction of facilities to house it will cost an additional $70,000.00, bringing the total project to approximately $165,000.00, of which half has been obtained.

Dr. J. M. Winkfield,

Strasburg, has been elected vice-president of the Board of Directors of the Massanutten Bank.

Dr. Frederick E. Vultee, Jr.,

Has been named chairman of the department of physical medicine and rehabilitation and director of the school of medicine at the Medical College of Virginia. He is now associate director of the Rehabilitation Institute of Chicago and will assume his duties in Richmond on the 15th of July. Dr. Vultee was formerly associate professor of physical medicine and rehabilitation at the College.

Plastic Surgery Program at University.

The plastic surgery program at the University of Virginia School of Medicine has been approved for

residency training by the Tri-Partite Committee of the American Medical Association. This accreditation marks the founding of the first and only approved program of its kind in Virginia. It will enable surgeons to receive thorough training in this specialty, and at the completion of the two-year program, qualify for the American Board of Plastic Surgery examinations.

The division of plastic and maxillofacial surgery was established at the University in 1956 by Dr. Claude C. Coleman, Jr. He is associate professor of plastic surgery and chief of the division and is also director of the Head and Neck Tumor Clinic, which he established in 1958.

Hand-Schüller-Christian Disease.

The cooperation of physicians is requested in a study of eosinophilic xanthomatous granulomatosis, and most specifically Hand-Schüller-Christian disease, being conducted by the Radiation Branch of the National Cancer Institute in the Clinical Center of the National Institutes of Health, Bethesda, Maryland. This study has as its primary purpose a search for therapeutic methods which may favorably affect the course of the disease.

Patients appropriate for this study are those with a histopathological diagnosis consistent with some phase of esoinophilic xanthomatous granulomatosis who have active disease. It is preferable, of course, that such patients not be seriously ill, as this precludes long term studies of effects. In order to minimize the effects of prior treatment, those patients who have not received antifolic-acid, radiation or steroid therapy for at least one month prior to their evaluation shall be considered most appropriate but previously treated patients will be considered for admission.

Physicians interested in the possibility of referring such patients should write or telephone: Dr. Charles G. Zubrod, Clinical Director, National Cancer Institute, Bethesda 14, Maryland (OLiver 6-4000, Ext. 4346); or Dr. J. Robert Andrews, Chief, Radiation Branch, National Cancer Institute, Bethesda 14, Maryland (OLiver 6-4000, Ext. 3351).

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Obituaries . . . .

Dr. William Meyer,

Prominent physician of Herndon, died following a heart attack on April 5th. He was sixty-eight years of age and a graduate of the Medical College of Virginia in 1918. Dr. Meyer began his practice at Messick and moved to Herndon in 1924. He was active in the civic affairs of his community, having served three terms on the Herndon Town Council, had been a director of the Citizens National Bank, was past president of the Rotary Club and an honorary member of the Arlington Kiwanis Club. Dr. Meyer was a 33rd degree Mason, a past potentate of Kena Temple, and a member of the Virginia Consistory. He held many other offices in the Masons and was well-known in Northern Virginia and throughout the shrine of North America.

Dr. Meyer was twice president of the Fairfax County Medical Society and was an associate member of the District and Arlington Medical Societies. He had been a member of The Medical Society of Virginia for thirty-seven years.

His wife, two sisters and four brothers survive him.

Dr. Churchill Robertson,

Salem, died May 4th, at the age of sixty-two. He graduated from the Medical College of Virginia in 1923. Dr. Robertson had practiced in Roanoke for more than thirty years before his retirement six years ago. He was a member of The Medical Society of Virginia, having joined in 1933.

Dr. Robertson is survived by his wife and two daughters. A brother is Senator A. Willis Robertson of Lexington.

Dr. Friedman.

In the death of Dr. Louis Friedman on February 16, 1960, of coronary thrombosis, this Society and its members lost a good and respected colleague. At the same time, our community suffered the loss of an able practitioner, one who had served well the needs of its people for forty-five years.

Dr. Friedman was born in Lithuania on December 15, 1888. He came to this country at the age of sixteen; and in Baltimore, studied at Deichman Preparatory School, from which he graduated in 1908. He received his Doctorate in Medicine in 1912 at the College of Physicians and Surgeons, which later was incorporated into the University of Maryland. He interned at the Sinai Hospital of Baltimore, and practiced general medicine for two years in that city, before entering the private practice of medicine in Norfolk in 1915.

For many years, he was a senior attending physician at Norfolk General Hospital, and active on its clinic staff. He was a staff member, also, at De Paul and Leigh Memorial Hospitals.

He was a member of this Society, The Medical Society of Virginia, and the American Medical Association.

Many, but not all of his colleagues, were aware of Dr. Friedman's intellectual attainments beyond the field of medicine. He was a devoted and capable student of ancient literature, and spent much of his leisure time in research in this field; especially in its relation to the history and philosophy of medicine.

In the passing of Dr. Louis Friedman from our scene, the Society has experienced the loss of a good friend and co-worker. We extend to Mrs. Friedman and her sons our deepest sympathy in their bereavement.

BE IT RESOLVED, then, that this expression of our feeling be read into the minutes of the Norfolk County Medical Society; that a copy be transmitted to Mrs. Friedman; and, further, that a copy be forwarded to the Virginia Medical Monthly for publication.

C. LYDON HARRELL, M.D., *Chairman*
ROBERT D. GLASSER, M.D.
BERNARD LIDMAN, M.D.

Dr. Smith.

The Mid-Tidewater Medical Society on February 26, 1960, lost by the death of Dr. James Smith one of its most loyal members, one of its founders and its first president.

Dr. Smith's faithful attendance on the meetings of the Society, at which he contributed sound counsel and pertinent comments wil be sorely missed. His versatile interests and keen mind kept him abreast of the rapidly changing field of medical practice in a truly commendable fashion.

Forty years ago, Dr. Smith took practical steps in community wide prophylactic immunizations when these measures were generally unknown in rural areas and he continued through his whole career progressive in thought and deed. His kindness and skill will be long remembered in Gloucester County where he served the people for more than half a century.

We feel a deep sense of loss in his passing and desire to extend to his family and friends our sympathy.

THEREFORE BE IT RESOLVED, that a copy of these memorials be spread on the minutes of the Mid-Tidewater Society, a copy be sent to the Virginia Medical Monthly, and a copy be sent to his family.

Adopted at a regular meeting of the Mid-Tidewater Medical Society on July 26, 1960.

M. H. HARRIS, M.D., *Secretary*

Dr. Lawrence.

On Sunday, March 13, 1960, in the company of his wife and Dr. and Mrs. Reuben Simms, Dr. Bruce Lawrence drove to his cottage on the Rappahannock River, to check for possible winter damage or other trouble. On entering the cottage he noticed severe chest pain, which increased rapidly and was self-diagnosed as a coronary thrombosis. In spite of emergency treatment, Dr. Lawrence passed away in a short time.

Bruce Lawrence was born in Richmond, on October 17, 1899. His father was Alonzo Robert Lawrence and his mother was Julia Lechler Lawrence. He attended the Richmond public schools and took his premedical education at the University of Richmond. He graduated in 1923 from Hahnemann Medical School, Philadelphia, and served his internship at Hahnemann Hospital there.

In July 1924 he began the general practice of medicine in Richmond. For many years he was associated with Retreat for the Sick Hospital in the capacity of anesthetist.

Dr. Lawrence was active in the Richmond Academy of General Practice from the time of its inception, and in 1959 he served as its President. He was also active in the Virginia Academy of General Practice, having served on the Board of Directors and on numerous committees. He was a member of the Richmond Academy of Medicine, The Medical Society of Virginia, the American Medical Association, and the American Academy of General Practice.

For ten years prior to June, 1959, he served on the State Board of Medical Examiners.

He was a member of the staff of the Richmond Memorial Hospital and Retreat for the Sick Hospital; of the courtesy staff of Stuart Circle and St. Luke's Hospitals.

Bruce Lawrence was a quiet, unassuming, dedicated and friendly man, who instilled confidence and affection in his patients. Few doctors have been more beloved by those whom they have treated. He was loyal to his friends, and though strong in his convictions, he was always ready to hear both sides of a question. He was wise in his judgment, patient, kind, compassionate and tolerant. He frequently would forego pleasure or vacation when he felt a sick patient might need him.

The Richmond Academy of Medicine realizes that it has lost a capable and valued member and friend, and extends to his wife and family its deepest sympathy.

The committee proposes that this resolution be adopted and incorporated in the minutes of the Richmond Academy of Medicine, and that a copy of this resolution be sent to his widow and to the Virginia Medical Monthly.

WILLIAM A. YOUNG, M.D.
REUBEN SIMMS, M.D.
RUSSELL G. MCALLISTER, M.D., *Chairman*

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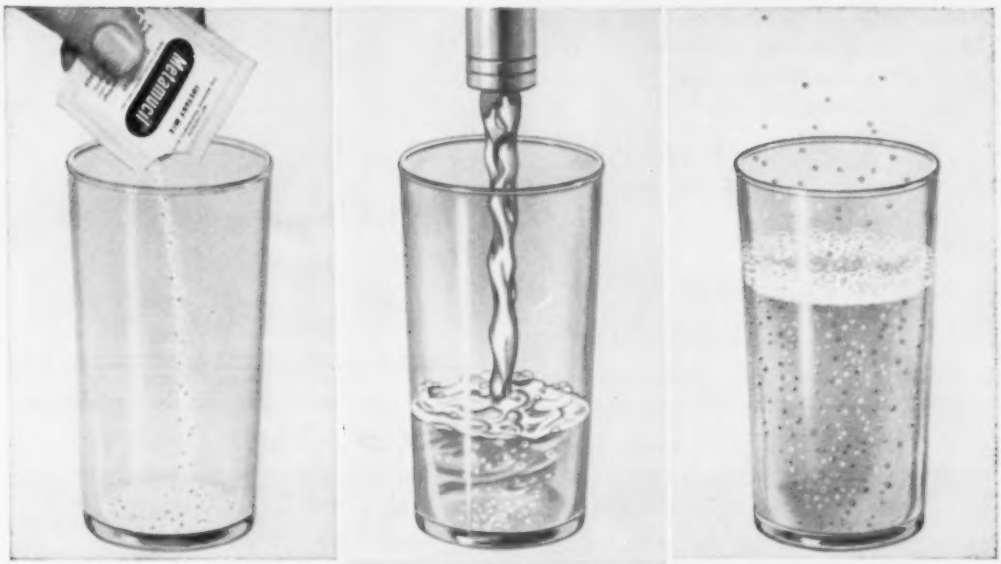

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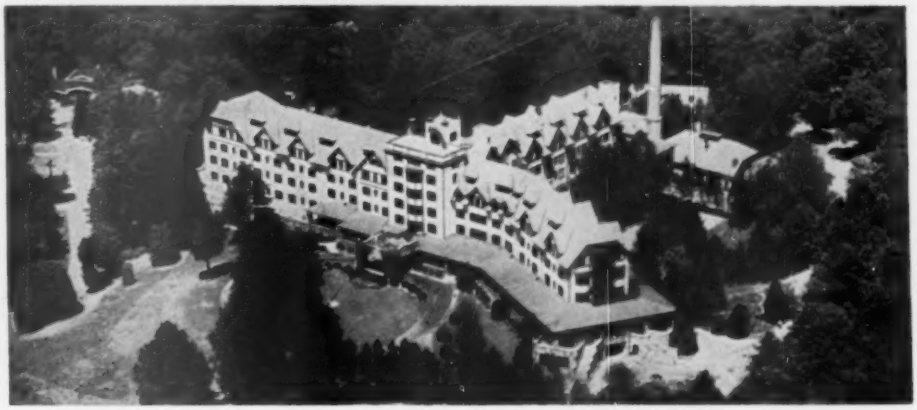

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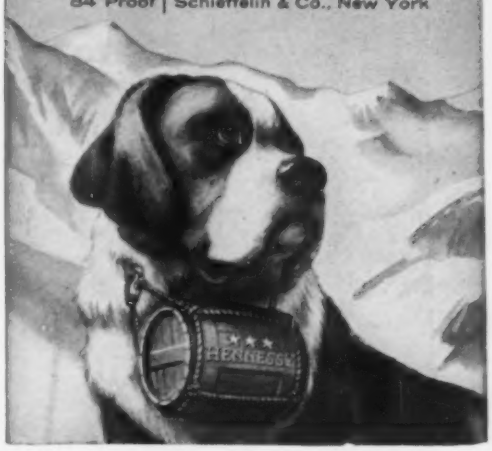

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For geriatric and chronically ill patients— a smooth, balanced action that lifts depression as it calms anxiety...rapidly and safely

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Dosage: Usual starting dose is 1 tablet q.i.d. When necessary, this may be gradually increased up to 3 tablets q.i.d.
Composition: 1 mg. 2-diethylaminoethyl benzilate hydrochloride (benactyzine HCl) and 400 mg. meprobamate.
Supplied: Bottles of 50 light-pink, scored tablets. Write for literature and samples.

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Same formula, plus

Neomycin sulfate	300 mg.

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Recent JAMA editorial statement clarifies the current controversy about dietary fats

Excerpted from the March 12, 1960, issue of The Journal of The American Medical Association:

"It is accepted generally that specific alteration in the diet will lower the concentration of cholesterol in the blood. The most effective results to date have been achieved by increasing consumption of polyunsaturated fatty acids, particularly linoleic acid. However, indefinitive and conflicting information has left much to the imagination of some food processors. Some of the largest vegetable oil processors in the United States have implied in advertisements that the cholesterol level can be lowered merely by adding polyunsaturated fatty acids to the diet. This selling campaign has created confusion among lay people, making it increasingly important that the physician clarify for his patients the conditions under which changes in the diet will be effective.

The patient should understand that if he increases his consumption of polyunsaturated fatty acids without reducing his intake of other fats, little is gained save for additional calories which could lead to obesity. A particular regimen will be effective only if polyunsaturated fatty acids are responsible for an appreciable percentage of the total fat calories. That is, they must replace rather than supplement some of the saturated fats and oils already in the diet.

* * *

Some manufacturers cite the "iodine number" of a fat or oil as evidence of the unsaturated fatty acid content of their product. This number is not a reliable indicator of therapeutic value because it measures monounsaturated and polyunsaturated fatty-acid content at the same time. A monounsaturated acid, like oleic, takes up two iodine atoms but does not affect the cholesterol concentration of the blood. A polyunsaturated acid, like linoleic, takes up four iodine atoms. In a product containing large amounts of oleic acid and small amounts of linoleic acid, the iodine number is nearly the same as it would be for a product containing little oleic acid and a modest amount of linoleic acid. Cottonseed oil has an iodine number of 110 and corn oil a number of 127; yet they each have about the same amount of linoleic acid.

* * *

Low-fat diets will not reduce the concentration of circulating cholesterol and lipoproteins as effectively as will diets containing an adequate percentage of polyunsaturated fatty acids. Weight-reduction regimens are basically low in fat, and if a lowered cholesterol level is necessary, planning must be done to maintain the proper ratio of saturated to unsaturated fats."

* * *

Herbert Pollack, M.D.
Associate Professor of Clinical Medicine
Postgraduate Medical School
New York University, New York

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Total unsaturated	70-75%
Palmitic, stearic and myristic glycerides (saturated)	25-30%
Phytosterol (predominantly beta sitosterol)	0.3-0.5%
Total tocopherols	0.09-0.12%

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LOW BACK PAIN

Trancopal®

A TRUE "TRANQUILAXANT"

relaxes skeletal muscle spasm

Trancopal®

BRAND OF CHLORMEZANONE

relieves
the pain
and disability
of
musculoskeletal
disorders

When enthusiastic gardening – or any of a host of other pleasant summer activities – brings on low back pain associated with skeletal muscle spasm, your patient need not be disabled or even uncomfortable for any length of time. The spasm can be relaxed with Trancopal, and relief of pain and disability follows promptly. The patient can usually continue his normal activities while taking Trancopal.

Lichtman[1,2] used Trancopal to treat patients with low back pain, stiff neck, bursitis, rheumatoid arthritis, osteoarthritis, trauma and postoperative muscle spasm. He noted that Trancopal brought satisfactory relief to 817 of 879 patients (excellent in 268, good in 448, fair in 101). "Chlormethazanone [Trancopal] not only relieved painful muscle spasm, but allowed the patients to resume their normal activities with no interference in performance of either manual or intellectual tasks."[1]

Gruenberg[3] also prescribed Trancopal for 70 patients with low back pain and observed that it brought marked improvement to all of them. "In addition to relieving spasm and pain, with subsequent improvement in movement and function, Trancopal reduced restlessness and irritability in a number of patients."[3] In another series of 193 patients Kearney[4] obtained relief with Trancopal in 181 patients suffering from low back pain and other forms of musculoskeletal spasm.

Trancopal enables the anxious patient to work or play. According to Gruenberg, "In addition to relieving muscle spasm in a variety of musculoskeletal and neurologic conditions, Trancopal also exerts a marked tranquilizing action in anxiety and tension states."[3] Lichtman[1] found that his patients in anxiety and tension states ". . . were in many instances able to continue their normal activities where previously they had been considerably restricted in their activities."[1] ". . . Trancopal is the most effective oral skeletal muscle relaxant and mild tranquilizer currently available." (Kearney)[4]

Side effects are rare and mild. "Trancopal is exceptionally safe for clinical use."[3] In the 70 patients with low back pain treated by Gruenberg,[3] the only side effect noted was a mild nausea which occurred in 2 patients. In Lichtman's group, "No patient discontinued chlormethazanone [Trancopal] because of intolerance."[1]

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Trancopal

potent muscle relaxant

effective tranquilizer

- In musculoskeletal disorders, effective in 91 per cent of patients.[5]
- In anxiety and tension states, effective in 89 per cent of patients.[5]
- Low incidence of side effects (2.3 per cent of patients).
  Blood pressure, pulse rate, respiration and digestive processes are unaffected by therapeutic dosage. It does not affect the hematopoietic system or liver and kidney function.
- No gastric irritation. Can be taken before meals.
- No clouding of consciousness, no euphoria or depression.

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Low back pain (lumbago)
Neck pain (torticollis)
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Fibrositis
Myositis
Ankle sprain, tennis elbow
Osteoarthritis
Rheumatoid arthritis
Disc syndrome
Postoperative muscle spasm

Psychogenic disorders
Dysmenorrhea
Premenstrual tension
Anxiety and tension states
Asthma
Angina pectoris
Alcoholism

How Supplied: Trancopal Caplets®

200 mg. (green colored, scored), bottles of 100.

100 mg. (peach colored, scored), bottles of 100.

Dosage: Adults, 200 or 100 mg. orally three or four times daily. Relief of symptoms occurs in from fifteen to thirty minutes and lasts from four to six hours.

References: 1. Lichtman, A. L.: *Kentucky Acad. Gen. Pract. J.* 4:28, Oct., 1958 • 2. Lichtman, A. L.: Scientific Exhibit, Internat. Coll. Surgeons, Jan. 4-7, 1959, Miami Beach, Fla. • 3. Gruenberg, F.: *Current Therap. Res.* 2:1, Jan., 1960 • 4. Kearney, R. D.: *Current Therap. Res.* 2:127, April, 1960 • 5. Collective Study, Department of Medical Research, Winthrop Laboratories.

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Component and Other Medical Societies in Virginia

(Officers and Others are Requested to Notify the **Monthly** of Changes)

SOCIETY	PRESIDENT	SECRETARY	TIME OF MEETING
Accomack County	D. F. Fletcher, Jr., Horsey	J. C. Doughty, Onancock	Quarterly
Albemarle County	G. S. Fitz-Hugh, Charlottesville	W. Copley McLean, Charlottesville	Monthly
Alexandria	B. C. Jones, Jr., Alexandria	W. J. Brennan, Alexandria	Monthly
Alleghany-Bath Counties	J. P. Hileman, Millboro	George N. Chucker, Clifton Forge	Bi-Monthly
Amherst-Nelson Counties	Lyddane Miller, Amherst	J. F. Thaxton, Tye River	Inactive
Arlington County	R. L. Norment, Arlington	Howard O. Mott, Arlington	Monthly
Augusta County	J. L. Davis, Waynesboro	Beverly Loesch, Waynesboro	5 times a year
Bedford County	S. L. Rucker, Moneta	O. B. Darden, Jr., Bedford	Quarterly
Botetourt County	M. S. Stinnett, Buchanan	E. L. Coffey, Buchanan	
Buchanan-Dickenson Counties	Robert Baxter, Grundy	J. S. Richardson, Grundy	
Charlotte County	Stuart Wilson Tuggle, Keysville	Thomas Watkins, Drakes Branch	
Culpeper County	J. B. Jones, Culpeper	James P. Baker, Culpeper	Monthly
Danville-Pittsylvania	J. W. Milam, Danville	G. B. Updike, Danville	Monthly
Fairfax County	J. E. O'Brien, Falls Church	Frances Ayres, Falls Church	Monthly
Fauquier County	E. H. Ashby, Jr., Remington	James L. Dellinger, Warrenton	Monthly
Floyd County	J. C. Rutrough, Willis	F. C. Bedsaul, Floyd	
Fourth District	Ray Moore, Jr., Farmville	J. D. Mason, Jr., Petersburg	Quarterly
Fredericksburg	L. F. Moss, Fredericksburg	J. E. Grimes, Fredericksburg	Quarterly
Halifax County	G. L. Wilkinson, South Boston	L. W. Roberts, South Boston	
Hampton	Helen Dorsey, Hampton	L. M. Lieberman, Hampton	5 times a year
Hanover County	J. D. Hamner, Jr., Ashland	Claude K. Kelly, Mechanicsville	
Hopewell	C. H. Dougherty, Hopewell	W. P. Youngblood, Hopewell	Monthly
James River	W. A. Pennington, Buckingham	J. H. Yeatman, Fork Union	Quarterly
Lee County	B. H. Owens, Rose Hill	H. A. Kinser, Pennington Gap	Quarterly
Loudoun County	S. S. Morrison, Leesburg	J. W. Gibson, Middleburg	
Louisa County	Griffith Daniel, Louisa	A. R. Southall, Louisa	Only call meetings
Lynchburg	G. B. Craddock, Lynchburg	L. F. Somers, Lynchburg	Monthly
Medical Society of Virginia	Allen Barker, Roanoke	R. I. Howard, Richmond	Oct. 9-12, Va. Beach
Mid-Tidewater	A. W. Lewis, Jr., Aylett	M. H. Harris, West Point	Quarterly
Newport News	J. Q. Hatten, Newport News	A. C. Stanton, Newport News	Monthly except June & July
Norfolk County	W. W. Taylor, Jr., Norfolk	Meyer Krischer, Norfolk	Monthly
Northampton County	W. F. Bernart, Nassawadox	W. S. Burton, Nassawadox	Monthly
Northern Neck	Paul Pearson, Warsaw	Harvey Goode, Jr., Kilmarnock	
Northern Virginia	Bradford Bennett, Winchester	Robert York, Berryville	
Orange County	H. C. McCoy, Gordonsville	R. S. LeGarde, Orange	Monthly
Patrick-Henry Counties	H. H. Price, Martinsville	E. T. McNamee, Stuart	Quarterly
Portsmouth	T. E. Jones, Portsmouth	M. C. Glynn, Jr., Portsmouth	Monthly except July & August
Princess Anne County	Fitzhugh Mayo, Va. Beach	A. B. Frazier, Virginia Beach	Monthly
Prince William County	Alvin Connor, Manassas	M. L. Nafsinger, Woodbridge	Indefinite
Richmond	R. V. Terrell, Richmond	Patty Boatwright, Richmond	Semi-Monthly
Roanoke	W. H. Kaufman, Roanoke	J. G. Snead, Roanoke	Monthly
Rockbridge County	John Yaeger, Lexington	K. J. Fox, Fairfield	
Rockingham County	H. P. Deyerle, Harrisonburg	C. E. Craun, Harrisonburg	Quarterly
Russell County	R. F. Gillespie, Lebanon	W. A. Davis, Dante	
Scott County	W. L. Griggs, Jr., Gate City	G. C. Honeycutt, Jr., Gate City	
Smyth County	C. O. Finne, Saltville	C. G. Thompson, Marion	Quarterly
Southwestern Virginia	J. G. Cox, Hillsville	M. A. Johnson, III, Roanoke	Semi-annually
Tazewell County	W. D. Damron, Richlands	Rufus Brittain, Tazewell	Bi-monthly
Tri-County	J. W. Lambdin, Franklin	Irwin H. McNeely, Franklin	Quarterly
Va. Acad. Gen. Practice	Boyd Payne, Staunton	S. F. Driver, Roanoke	May 11-14, 1961
Am. Col. Chest Phys.	G. C. Pearson, Blue Ridge	C. C. Smith, Catawba	October
Va. Sec. Amer. Col. Phys.	William Parson, Charlottesville	William H. Harris, Jr., Richmond	Twice a year
Va. Diabetes Assoc.	W. R. Jordan, Richmond	L. B. Sheppard, Richmond	Twice a year
Va. Ob-Gyn. Soc.	W. D. Suggs, Richmond	Brock D. Jones, Jr., Norfolk	Twice a year
Va. Orthopedic Soc.	Wm. Deyerle, Richmond	F. B. Carpenter, Richmond	Twice a year
Va. Pediatric Soc.	O. W. Booth, Newport News	G. G. Arnold, Richmond	Twice a year
Va. Peninsula Acad. Medicine	G. S. Grier, III, Newport News	T. W. Sale, Hampton	Monthly
Va. Neuropsychiatric Soc.	Frederick Woodson, Norfolk	Howard Ashbury, Staunton	Semi-annually
Va. Radiological Soc.	C. D. Smith, Roanoke	F. A. Kearney, Hampton	Twice a year
Va. Soc. Anesthesiology	Patricia Andrews, Charlottesville	Campbell Harris, Jr., Richmond	Semi-annually
Va. Soc. Internal Med.	M. M. Pinckney, Richmond	T. N. Hunnicutt, Jr., Newport News	Twice a year
Va. Soc. of O. L. & O.	M. P. Smith, Richmond	M. K. Humphries, Jr., Charlottesville	Twice a year
Va. Soc. of Pathology	David Smith, Charlottesville	George J. Carroll, Suffolk	Quarterly
Va. Soc. Plastic & Reconstr. Surg.	C. C. Coleman, Jr., Charlottesville	Leroy Smith, Richmond	
Va. Surgical Soc.	B. W. Rawles, Jr., Richmond	Carrington Williams, Jr., Richmond	Yearly in Spring
Va. Urological Soc.	Ralph Landes, Danville	G. W. Link, Petersburg	October
Washington County	Harry Hayter, Abingdon	J. C. Placak, Abingdon	Monthly
Williamsburg-James City	B. H. Bailey, Yorktown	B. I. Bell, Jr., Williamsburg	
Wise County	G. C. Snead, Derby	William Schmidt, Norton	
Wythe-Bland Counties	W. R. Chitwood, Wytheville	C. B. Hughes, Wytheville	Quarterly

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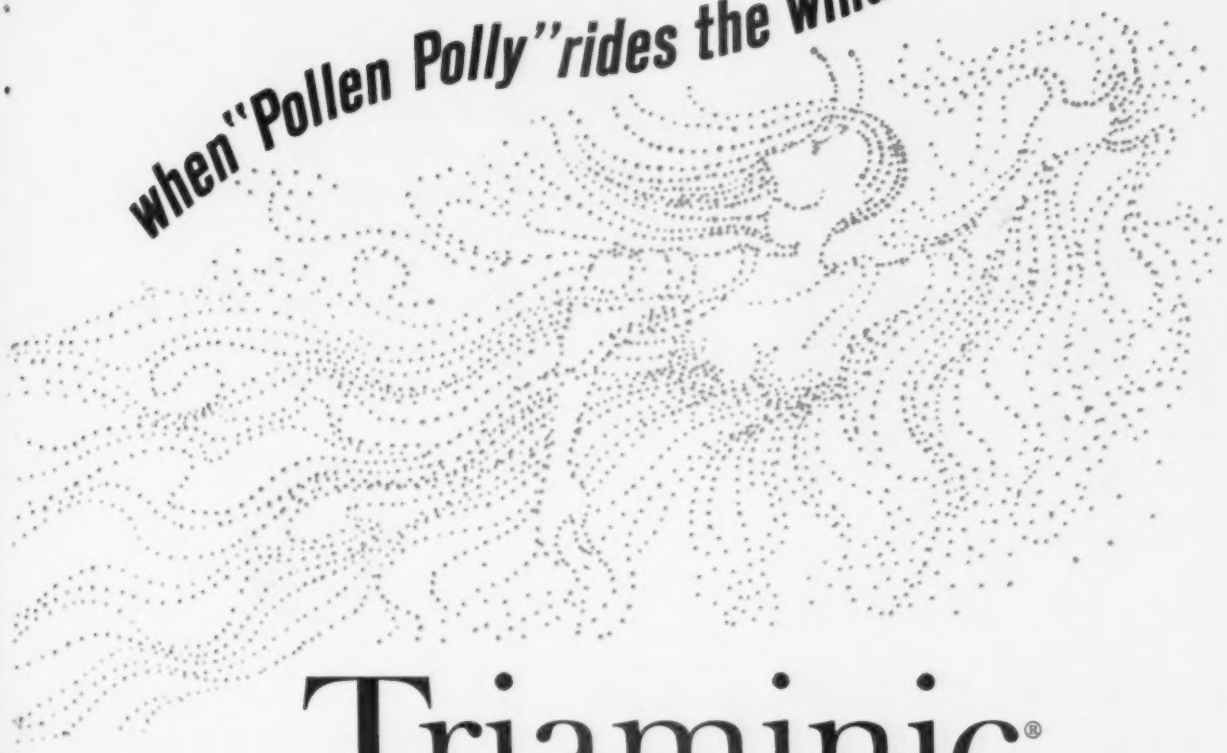

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TRIAMINIC JUVELETS® ½ the formulation of the Triaminic Tablet with timed-release action.

TRIAMINIC SYRUP each teaspoonful (5 ml.) provides ¼ the formulation of the Triaminic Tablet.

References: 1. Fabricant, N. D.: E. E. N. T. Monthly **37**:460 (July) 1958. 2. Lhotka, F. M.: Illinois M. J. **112**:259 (Dec.) 1957. 3. Farmer, D. F.: Clin. Med. **5**:1183 (Sept.) 1958. 4. Fuchs, M.; Bodi, T.; Mallen, S. R.; Hernando, L., and Moyer, J. H.: Antibiotic Med. & Clin. Ther. **7**:37 (Jan.) 1960. 5. Halpern, S. R., and Rabinowitz, H.: Ann. Allergy **18**:36 (Jan.) 1960.

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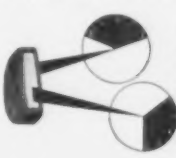

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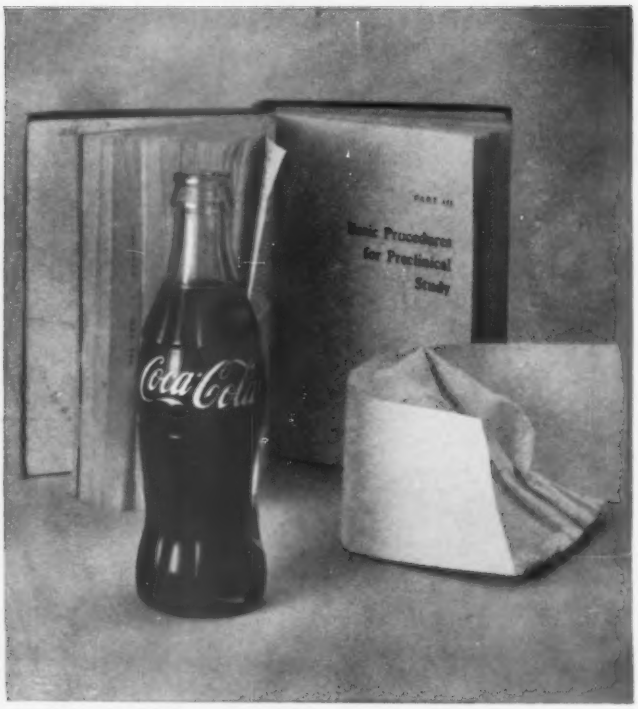

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The State Board of Medical Examiners of Virginia

The next meeting of the Virginia Board of Medical Examiners will be held in the John Marshall Hotel, Richmond, Virginia June 13, 1960. The examination will be held in the same hotel June 14-17 inclusive.

All applications and other documents pertaining to the examination or to matters to be discussed by the Board must be on file in the Secretary's office on or before May 20, 1960. The Secretary of the Board is Dr. K. D. Graves, 631 1st Street, S.W., Roanoke, Virginia.

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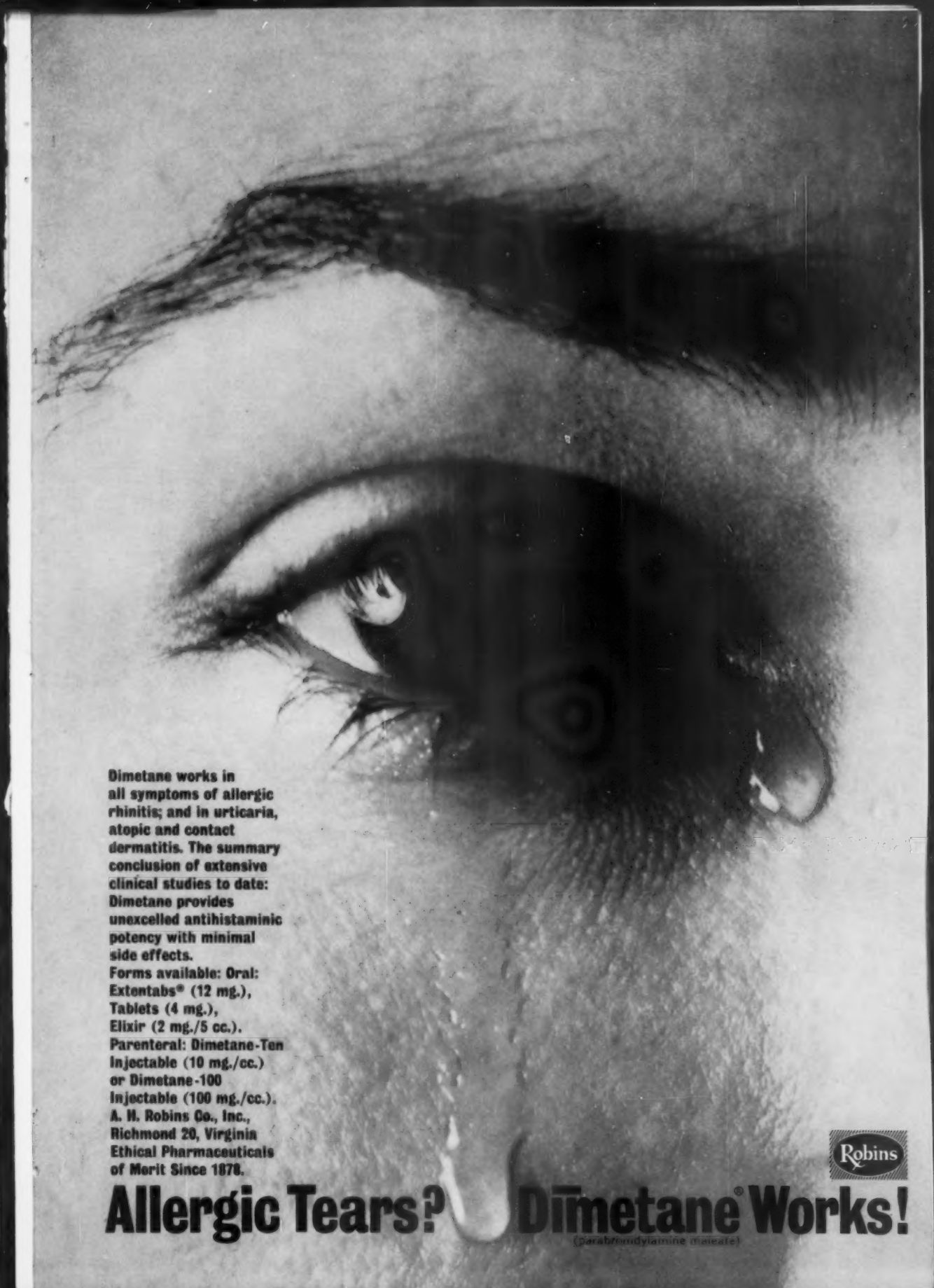
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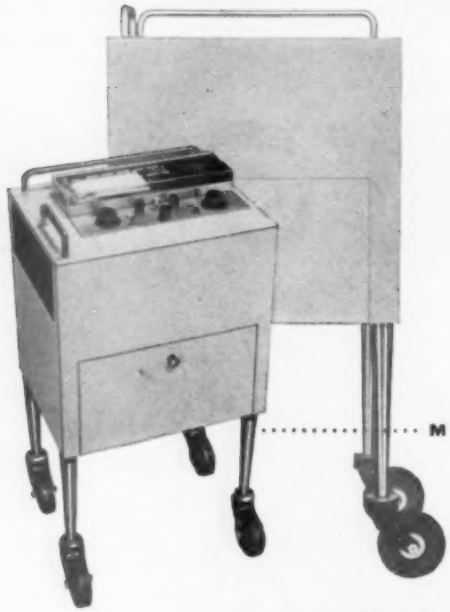

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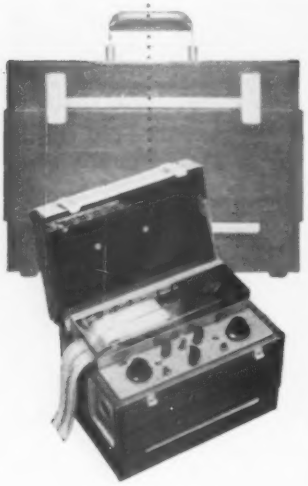

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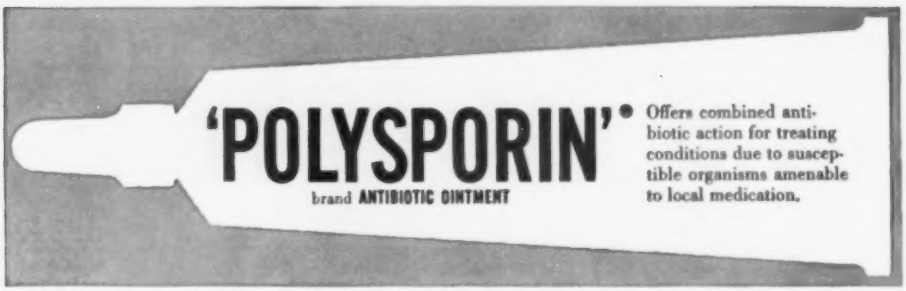
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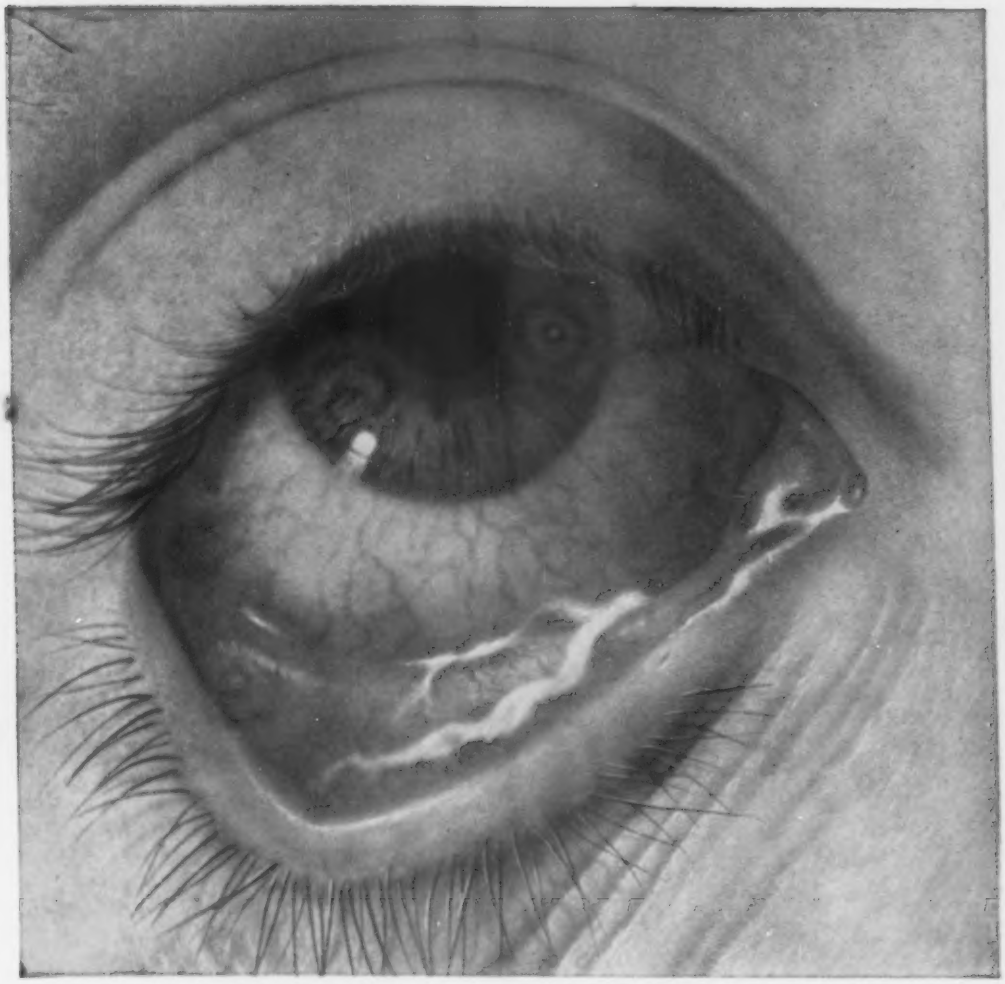

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1. Lippmann, O.: Arch. Ophth. **57**:339, March 1957.
2. Gordon, D.M.: Am. J. Ophth. **46**:740, November 1958.

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REFERENCES: 1. Yow, E. M.: Practitioner 182:759, 1959. 2. Yow, M. D., and Womack, G. K.: Ann. N. Y. Acad. Sci. 76:363, 1958. 3. Bunn, P. A., Baltch, A., and Krajnyak, O.: Ibid. 76:109, 1958. 4. Council on Drugs, J.A.M.A. 172:699, 1960.

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INDEX TO ADVERTISERS

American Geloso Electronics, Inc. 11
Ames Company, Inc. ..... Inside Back Cover
Appalachian Hall 46
Arnar Stone Lab., Inc. 57
Brayten 13
Bristol 14-15, 78-79
Burroughs-Wellcome Co. 16, 76
Ciba 23, 52-53
Coca-Cola 70
Component Societies 67
Davies, Rose & Company, Limited 53
Desitin Chemical Co. 40
Dyer, David A., Insurance 12
Eli Lilly and Company 42
Gill Memorial Eye, Ear and Throat Hospital, Inc. 46
Geigy 38
Jefferson, A. G. 56
Johnston-Willis Hospital 44
Jones and Vaughan 7
Lederle 24-25, 36, 44, 51, 56, 80, 81
Lorillard 8
Mayrand, Inc. 10
Medicine in Virgina 56, 70
Merck Sharp & Dohme 5, 77
Parke, Davis & Company ..... Second Cover-2
People's Service Drug Stores 62
Physician's Products Co. 39
Pitman-Moore Co. 82
Plyler's Nursing Home, Mrs. 48
Richmond Eye Hospital—Richmond Ear, Nose and Throat Hospital 45
Richmond Hotels Incorporated 56
Riverside Convalescent Home 45
Robins, A. H. Company 34-35, 59, 73
Roerig 26, 75
Rucker, Marvin Pierce 80
Saint Albans 48
Sanborn Company 74
Sardeau, Inc. 22
Saunders, W. B., Company 9
Schering 6, 28-29, 41, 71
Schieffeln & Co. 51
Searle 43
Smith-Dorsey 68-69
Smith Kline & French Laboratories ..... Back Cover
Squibb 17
St. Elizabeth's Hospital 47
St. Luke's Hospital 50
St. Paul-Western Insurance Companies 54
State Board of Medical Examiners, The 70
Stuart Circle Hospital 47
Terrace Hill Nursing Home 44
Today's Health 70
Tucker Hospital, Inc. 50
United States Brewers Foundation 58
U. S. Vitamin Corp. 32-33
Vale Chemical Co. 31, 72
Valentine Company, Inc. 51
Wallace Laboratories 18-21, 27, 37, 55
Wesson Oil 60-61
White Cross Hospital 49
Williams Printing Co. 54
Winthrop Laboratories 30, 63-66,

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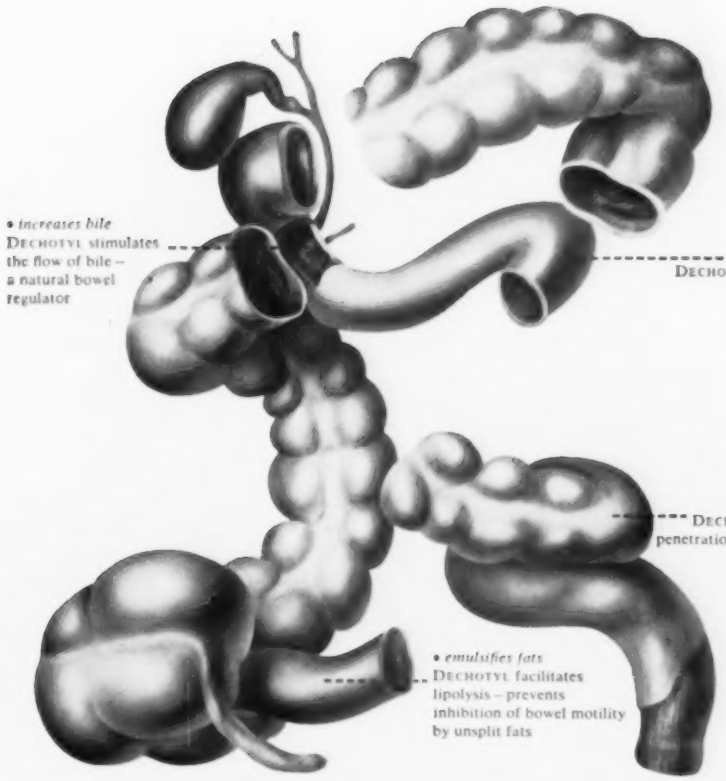
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